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CONTRIBUTIONS TO ABDOMINAL SURGERY

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CONTRIBUTIONS TO
ABDOMINAL SURGERY

BY THE LATE

HAROLD LESLIE BARNARD, M.S., F.R.C.S.

SURGEON (WITH CHARGE OF OUT-PATIENTS) TO THE LONDON HOSPITAL

EDITED BY

JAMES SHERREN, F.R.C.S.

SURGEON (WITH CHARGE OF OUT-PATIENTS) TO THE LONDON HOSPITAL; SURGEON TO THE POPLAR HOSPITAL FOR ACCIDENTS, ETC.

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PREFACE

BARNARD'S premature death is a loss to Surgery. From the commencement of his career on the Staff of the London Hospital, he was attracted to Abdominal Surgery, and his more important communications to various societies and to the medical papers dealt with this. His last work, on Subphrenic Abscess, published the year of his death, was universally recognized as a great advance on previous knowledge, and has done much to clarify our ideas on what is one of the most difficult subjects in this branch of Surgery; careful perusal of this work, putting into practice its teachings, will be the means of saving many lives.

Barnard had been working for several years at the subject of Intestinal Obstruction, and had planned an exhaustive treatise. The first-fruits of his work were his articles on Gall-stone Obstruction in the *Annals of Surgery*, embodied in his later work on this subject, and his lectures on Intestinal Obstruction at the Medical Graduates' College and Polyclinic. The book was put on one side while he was engaged on his article for Allbutt and Rolleston's "System of Medicine." It is only necessary to glance at the work to see what a tremendous amount of labour it involved; it is a mine of information and of personal observation.

A small committee, consisting of Mr. Hugh Barnard, Professor Arthur Keith, Dr. Cecil Wall, and myself, met on Harold Barnard's death to discuss the best form a memorial should take. We considered we should be carrying out the desires of our friend and colleague best by attempting, however imperfectly, to fulfil in some degree the work he had planned and delighted in. I was given the privilege of editing this work.

On going through the mass of notes and the number of illustrations left, I found that there were considerable portions of the subject of Intestinal Obstruction that had never been published. After Messrs. Macmillan had kindly given their permission to use as much of Barnard's article from Allbutt's "System of Medicine" as we needed, it was decided to incorporate new material with this, and to illustrate, as far as possible, from the drawings Barnard had collected for his larger work.

The sections on Fæcal Obstruction and on Congenital Dilatation of the Colon have not been previously published; those relating to Gall-stone Obstruction, Strangulation by Bands and by Meckel's Diverticulum, and to Obstruction by Foreign Bodies contain much new material. In addition, his articles on Appendicitis are reprinted by kind permission of the editors of the *Lancet* and *Clinical Journal*, to whom our best thanks are due. The volume concludes with Barnard's last contribution to abdominal surgery, with which his name will always be associated; for permission to reprint this we thank the editor of the *British Medical Journal.*

In no sense have any of the articles been "edited." My task has been to incorporate and illustrate, not to revise. No alteration has been made in the wording. Those who had the privilege and pleasure of listening to Barnard's teaching will recognize his inimitable methods.

In conclusion, the committee would tender their grateful thanks to Messrs. Macmillan for the kind permission, so readily given, which made this volume possible.

JAMES SHERREN.

January, 1910.

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HAROLD LESLIE BARNARD

(Died August 13, 1908)

I.

To those of us who had passed, reluctantly enough, from our places at the London Hospital, the news of Barnard's death was difficult of belief. Even though we had failed to tread the Whitechapel Road for a few months, or perhaps a year or two, the memory of him was of something so vital, so combative, if the word be rightly understood, that some error in the brief announcement of the newspapers seemed the only explanation. And yet, since at all times, and perhaps more readily at a hospital than in most places, such things are happening, we could only guess at last that behind the printed tidings lay the history, unknown to us, of some sudden calamity.

It was only when subsequent days revealed that the workers at his side, his everyday colleagues, even his family, had been taken as completely by surprise, that the real truth emerged. We learned then that there had been no sudden calamity at all; but instead a slow fight with death, carried on to its certain and foreknown conclusion, for more than a year, with a spirit so gay and intrepid as to deceive his nearest comrades, and to convert what seemed to most of us a very bitter and ironical tragedy into a shining triumph of human will.

Looking back, it says everything for Barnard's greatness that this silent struggle becomes characteristic. In a certain sense it is what we should have expected; and the desire for an equal courage is our sincerest homage to him. Confronted by many obstacles, a false start upon a commercial career, a lack of financial means, certain always abiding mannerisms, there were times when, as he once put it himself, there was a " slump in Barnard." He was big enough to live through them with a smile. Twelve months before he died he may be said to have crossed the threshold, if not of a general celebrity, at any rate of a permanent scientific regard. A tardy laurel or two had even fallen upon his brow, and for those of us who were onlookers his days of warfare had seemed to have been very nearly accomplished. It was then however that circumstance, giving him no truce, challenged him to his last fight, wherein he saw an inevitable defeat awaiting him. How serenely he picked up the glove is now known to us all. There was no abatement of enthusiasm even in the smallest issues of life, and he could find time, under the shadow of death, to lower his handicap at golf.

Much of the legacy that he has won for us through his strenuous years will be found between the covers of this volume. But more has escaped print ; and of this not the least significant portion is the high example of that last Promethean conflict.

For this good fight, and whatever may have been his initial endowment of vital energy, his will must have kept itself in constant training ; and a brief survey of his life's story seems to prove this very clearly. Born in January, 1868, upon the confines of Highbury, Harold Barnard was the great-nephew of Michael Faraday, and the second son of James Barnard, an engraver and designer in precious metals. In the days when Highbury was still the Ultima Thule of London's northward progress, the young Barnard accompanied his sisters across the fields to a pre-kindergarten establishment in the Petherton Road, continuing to attend there until, in January, 1879, at the age of eleven, he entered the Rev. George Vasey's private school in Highbury Park.

Even before this time he had made virgin sallies upon the secrets of science, and there are family witnesses to the unsuccessful and personally disastrous attempt at a too rapid admixture of concentrated sulphuric acid and water, and a more triumphant (but again temporarily disabling) project of lighting the dining-room gas with an electric spark. From the date of his entry at the private school however, both his militant and scientific faculties appear to have thriven vigorously. Never afraid of a minority at any stage of his career, at school he seems rather to have chosen one. Thus, if the enthusiasm of his fellow-scholars bubbled over for Oxford, in the days before the University boat-race, young Barnard would appear jauntily in the Cambridge colours, with two or three light blue comrades hovering, somewhat less confidently, in his wake. If the school were Conservative, Barnard led and protected in many a bloody combat a minute party of fellow-Radicals; and his prowess on these occasions would appear to have been at least respectable.

Side by side with this, and perhaps indeed an expression of the same quality, his leanings towards the natural sciences began at the same time to develop the propensities that afterwards became their chief strength. The spirit that forbade compromise on schoolboy-politics kept his intelligence alert at the tackling of textbooks. He would take few things for granted, and, in very young days indeed, made a habit of personally verifying, wherever this was possible, the statements of fact that were presented to him. An affection for the studies of anatomy and physiology would also seem to have awakened in him at this time; and there are still preserved some very carefully coloured drawings of the human blood-circulation system, and the bony skeletons of some birds' claws, dissected out, skilfully articulated, and pasted upon paper.

While this love for scientific research was rapidly becoming the dominating interest of his life, he had developed also a healthy devotion to athletics that never left him. Playing football for his school, he was equally fond of long-distance swimming and walking. And it was in walking that many, if not all, of his boyish holidays were spent—an arrangement encouraged by his father for reasons upon which an extract from the family diary may perhaps throw some light. Thus we read that in 1883 " Harold's and Evelyn's two trips to Cornwall and Salisbury, accomplished with so much ease and at so little cost, will probably lead to more extensive travels, as a knowledge of peoples and their countries, following close school study, helps to broaden ideas and to make capable men "—a quotation that may fittingly stand for a lantern upon the boy's home-life at this period.

Later on in this same year, and three months before his sixteenth birthday, Barnard and his brother were sent out to an uncle, who was then ranching in the State of Oregon; and it is perhaps typical of the very practical régime under which they were brought up that both boys, before sailing, had to pass the first examination of the St. John's Ambulance Association, while Harold, in addition, was made to promise that he would read, during his visit to America, for the London University matriculation. Oregon at fifteen—and the London matric! . . . one sighs a little that Romance should have needed quite so stern a policeman.

To the ten months spent upon his uncle's ranche Barnard always looked back with the keenest pleasure. Indeed, for all his good intentions and some rather painful efforts at fulfilment, the spectre of the coming examination seems to have faded satisfactorily from his horizon. It was the only one in which he ever failed. Hunting, shooting, trapping, playing with an axe, the life of which he then obtained a glimpse must have been extraordinarily attractive to him; and he seems to have revelled very thoroughly in its opportunities for adventure.

Describing one of these in a letter home, " the black bear," he tells us, " although a formidable animal, is not to be feared if resisted with courage "—a remark sententious enough to have been dear to his father's heart, and foreshadowing certain characteristics of speech and temperament that became familiar to his colleagues in later years. Looking back, one can only be sorry that these good days were so brief, and that no further chance of renewing them seems to have come into his life.

In 1884, having returned to Highbury and failed to pass his matriculating examination, Barnard entered a firm of wholesale timber-merchants with the intention of engaging in a commercial career; and his life during the following years seems to have resembled that of any average City clerk. Working a regular number of hours each day, he devoted his evenings to such recreation as he could afford, learned to play the violin, and dabbled in amateur theatricals. He was never happy in these new associations however, and before the end of his apprenticeship had definitely decided to abandon commerce as a life-work, and in the face of numerous difficulties to embark instead upon the study of medicine and surgery.

II.

Entering the London Hospital in the year 1888, at the age of twenty, Barnard seems from the first to have been thoroughly resolved that henceforward his new studies must fully monopolize his time. For to the natural attraction that they possessed for him was added the remembrance that the past three or four years had been to a certain degree misspent—a fact that united with the scantiness of his means to become both his spur and handicap in the struggle before him. Others, more softly circumstanced, might dally gently with scalpel and textbook, but for him at any rate, they must be sword and Bible, and no other interests must be allowed to dispute their supremacy.

For such a youth it would have been easier to prophesy a scholastic success than a general popularity, and when one finds the pages of his earlier notebooks headed with little platitudes concerning diligence and the nature of his studies, it is hard to see how he could have avoided a certain active and perhaps healthy hostility. The product of a suburban private school, a somewhat trite, if entirely loving, up-bringing, and the atmosphere of a merchant's office, there would perhaps necessarily have been some indefinable things that it would have been wonderful for him to possess. A gentler and less uncompromising personality might have bridged the lack of them, or acquired them at a lesser cost. Barnard, and how rightly it is a little difficult to say, seems never to have made up his mind if they were worth acquiring at all.

Yet some of them came to him, perhaps in spite of himself, in later days. And at the same time his generation, grown older in judgment, and perceiving at last the real man, discovered also the absolute importance of these matters to have dwindled a little. The unriper verdict of contemporary youth, fresh from school and University, was at once less kindly and discriminating. And Barnard was more sensitive than his observers may have believed. But if this should seem to spell unhappiness, the impression given would scarcely be a right one. For while he must often have been wounded by what was at all times obvious to him, it was in the mastery of difficulties that his chief joy always lay; and to be more or less alone in the fight can never have very deeply concerned him.

Academically he can have had but few disappointments. In his first year he obtained a scholarship in anatomy and physiology; in his second, scholarships in anatomy, physiology, and chemistry, and also a minor surgery prize and a prize for dissecting. In his third year he gained the Letheby Prize in chemistry, and in his fourth year he took a certificate in obstetrics, his double qualification of M.R.C.S., L.R.C.P., and two months later began his first hospital appointment as senior dresser.

Writing of these student days, his friend and contemporary, Dr. H. G. Felkin, tells us that "he worked quite unusually hard, and got through an astonishing amount in a short time. This was, no doubt, one reason why others saw so little of him. He was also, in his own way, very impulsive, and went for his object by the shortest and most direct route, and was inclined to disregard any side-issues. He read a good deal (one might more properly say 'devoured') outside his work, especially literature dealing with science generally and metaphysical and social problems. I often worked with him, especially at histology, and found him always the enthusiast, whatever the subject in hand might be, and used to admire his invariable mental freshness."

During a portion of his student career Barnard lived in one of the tenement houses near the hospital, sharing two rooms with one of his brothers; and in spite of occasional week-end holidays, during which, Dr. Felkin writes, "he used to go for long walks at a tremendous pace, much faster than pleased me," this combination of hard living and intense mental application took not infrequent toll of his physical health. Thus his brother tells, at this time, of more than one attack of fainting owing to sheer exhaustion, and Dr. Felkin writes of "frequent insomnia and headaches, though he always revived very quickly." Though often warned by his friends, and especially by the late Dr. James Anderson, he appears to have paid very little attention to their advice; and in the more advantageous circumstances opened to him by his qualification, his health seems to have improved again considerably.

Becoming Senior Dresser to Out-Patients on December 1, 1892, the next three years were almost entirely occupied in the holding of various resident appointments. He was Receiving-Room Officer and Ophthalmic Clinical Assistant in 1893, and House-Physician to Drs. Fenwick and Warner in 1894. Later in the same year he became House-Surgeon under Mr. Hurry Fenwick, who was particularly impressed with his ability. This last appointment concluded for a time his connection with the hospital, and in 1895, having taken his M.B. degree of London University in the first division, his Fellowship of the College of Surgeons, and his Bachelorship of Surgery (also in the first division) at the London University, he was appointed Demonstrator of Physiology under Dr. Leonard Hill, in the London Hospital Medical College—a post that he held till March, 1897. Here, under the guidance of Dr. Leonard Hill, he carried out researches on the influence of gravity on the circulation, and collaborated in the invention of a sphygmomanometer of considerable clinical value. Concerning their two years' co-operation, Dr. Leonard Hill wrote in the *British Medical Journal* of August, 1908, that "he had the highest scientific ability, and might have developed into a great scientific worker had opportunity offered itself. If he had been able to follow that line of life, he would have rendered signal service to the advance of pathology."

How catholic were his interests however, and how great his mental energy, may be gathered from the fact that during this time he was also working for the degree of Master of Surgery at the London University, which he took in December, 1896. In March, 1897, giving up his college appointment, he became Surgical Registrar to the hospital, a post that he held for two years; and in the following year, 1900, having already been made Surgical Tutor, he attained his first great ambition and the well-earned reward of twelve strenuous years in being appointed an Assistant Surgeon on the staff of his hospital.

III.

While it is as a surgeon that Barnard himself would ask to be judged, and while it is as a surgeon that upon the world outside his reputation was dawning, there is perhaps less need here to lay emphasis on this aspect of his life. To his acute observation, his imaginative insight, and enormous diligence in this capacity, these collected writings of his are the best monument. And though it is difficult to believe that they can be regarded as building anything but a broken column, richer in promise than accomplishment, yet the accomplishment itself, when one reflects that, after all, it represents merely the spare moments of less than eight crowded years, is one of which any man might be proud.

Nor was Barnard a mere man of theory and letters. A wise and practical surgeon, he was both a brilliant operator and an excellent diagnostician. Indeed in some respects he had already obtained as the latter a unique position. Thus, as a writer in the *British Medical Journal* quite justly observes, Barnard's knowledge of abdominal conditions was unrivalled in a man of his age. And his selection as the authority upon Intestinal Obstruction in Allbutt and Rolleston's "System of Medicine" may be taken as a public recognition of this. That his interests were not confined to this particular department of surgical science however the variety and extent of these collected papers make sufficiently clear. And both by performance and implication they prove to us, as nothing else could well do, how great has been his loss to British surgery.

As a friend and colleague, it is safe to say that, at the time of his death, Barnard had lived down any obloquy that may have survived from his student days. Liberal to a fault, and the possessor of a quite characteristic vein of humour, he entered thoroughly into the social life of his alma mater. He was president of the swimming club, vice-president of the football, boxing, fencing, and gymnastic clubs, and vice-president of the Medical Society. He was seldom absent from a cup-tie match. And, as his friend Dr. Wall has said for us, those who were present will not readily forget his entertainment of the victorious Rugger team that brought back the cup to the London after an absence of twenty years.

Lastly one turns to consider Barnard as a teacher; and it is characteristic that in this, man's most generous function, he was perhaps more excellent than in any. Unsparing of incompetence, and a little over-harsh at times to mere excusable ignorance, when he met these in the sphere of practical application, he brought to the class-room an unfailing good-temper and a very clear sense of the teacher's limitations.

With customary incisiveness he solved the problem that must beset every thinker turned instructor. As the one, his imagination, his powers of perception and judgment, his faculty for criticism, might well roam unharnessed. As the other, they must be driven on a firm rein. For his pupils' sakes, still subject to examining boards and to be judged by textbook standards, he must be trenchant, dogmatic, illuminative. And Barnard's mnemonics, fastened into memory with some typical anecdote, gathered impartially from the drawers of fact and fiction, must have piloted many of us through the dark moments of some crucial *viva*.

For whatever he may have been as a student, there can be no doubt at all about Barnard's popularity as a teacher—a popularity that was the response to a real genius in this respect, and one that never waned till he taught no more.

Indeed it is difficult now to picture any but crowded benches during one of his lectures; and looking back, with the fuller knowledge of the man that death has brought us, this becomes a pleasant thing to remember.

For Barnard enjoyed his lectures, and it may be accounted among a too slender list of visible rewards that he should have perceived his own enjoyment to have been communicable. Never failing to infuse his lectures with all his own energy of the hour, he allowed his personality to escape him, on these occasions, with admirable success. And as a result there were no lectures quite like them. Of literary style or polish they possessed nothing. Talking like a London schoolboy on a holiday, it was perhaps the schoolboy in his listeners that he succeeded most in rousing; and his lectures assumed in consequence a certain tempestuous quality that distinguished them among more sober brethren.

They bubbled along to an ever-varying tumult of scribblings, stampings, and roars of laughter. This rum sort of chap had been out all day (and more than half the night, if we had but known)—out with a net, looking for things. How far had he been? Oh never mind that just now. Look here, all sorts of queer things, the moths, butterflies, and beetles of fact that he had succeeded in collaring among the broad fields of human experience or groped for in the yet wider darknesses of philosophic surmise. Here they are, all neatly pinned down for you fellows to see. Any more where they came from? Oh lots, but you've got to look at these first. That's an odd one now, isn't it? Tell you a yarn about that one. . . .

Is it wonderful that few academic hours fled so quickly as these, or so surely achieved their end?

And perhaps, when all is said, the picture that will remain with most of us, when the apparent tragedy of his death has been forgotten, will be that of the Highbury schoolboy, scornful of best clothes and unconcerned to cultivate refinement, a little careless perhaps of the neighbours' hedges, but willing to go shares with anybody, faring out into life with box and net, and unalterably resolved, wherever he may find it, to go bird's-nesting up the tree of knowledge.

The flesh may have taken manhood upon itself, becoming stronger and more skilful, or growing weaker gained in tenderness and patience. But it is not the flesh that we shall remember. And the spirit that was in the schoolboy never died.

H. H. BASHFORD.

CONTRIBUTIONS TO ABDOMINAL SURGERY

INTESTINAL OBSTRUCTION

PART I.—THE GENERAL CONSIDERATION OF INTESTINAL OBSTRUCTION

Synopsis.—1. Definitions. 2. The general pathology of intestinal obstruction: (A) Morbid anatomy—(i.) of a case of chronic obstruction, (ii.) of a case of chronic obstruction which has become acute, (iii.) of a case of acute obstruction of normal intestine; (B) the general consideration of the symptoms of intestinal obstruction and their pathological explanation. 3. The general symptoms of intestinal obstruction—its three clinical varieties. 4. The diagnosis of intestinal obstruction: (i.) The general diagnosis, (ii.) the differential diagnosis of different causes, (iii.) the differential diagnosis from similar conditions. 5. Terminations, duration, and prognosis. 6. The general treatment.

1. Definitions.

Intestinal obstruction is a condition in which the intestinal contents are more or less hindered in their passage along the bowel. The mechanical and organic causes only of obstruction of the intestinal lumen will be dealt with here.

Ileus is an ancient term incapable of precise definition, and therefore should not be used. It is often employed to signify paralytic dilatation of the intestine, but has been applied to any form of abdominal distension, and is sometimes used as synonymous with intestinal obstruction. *Stenosis* is a narrowing of the lumen of the intestine. *Occlusion* is complete closure of the lumen of the intestine. *Obturation* of the intestine is that condition in which the lumen is blocked by a foreign body. *Stricture* is stenosis or occlusion of the lumen of the gut from some disease attacking an annular portion of its wall. *Constriction* is stenosis or occlusion of the intestinal lumen by some cause surrounding the gut. *Strangulation* refers to the condition of a coil of intestine when it is so constricted that its lumen is occluded, its vessels are obstructed so that, as a rule, intense venous hyperæmia is produced, and its nerves are vigorously excited.

2. THE GENERAL PATHOLOGY OF INTESTINAL OBSTRUCTION.

(A) **Morbid Anatomy.**—Intestinal obstruction has three distinct clinical forms: (i.) Acute intestinal obstruction, in which the normal intestine is suddenly and completely occluded, and, as a rule, some segment of the gut is strangulated. In such a case the changes in the strangled loop are perhaps the most important, but other changes are to be found in the segments of intestine above and below the obstruction. These changes are for the most part the result of the interruption of the intestinal stream, but so rapidly fatal are these cases that little time is allowed for structural alteration. (ii.) Chronic intestinal obstruction. There are many causes for this condition, but all agree in that the lumen of the intestine is narrowed, so that a partial obstruction is produced. The prolonged course of these cases allows well-marked anatomical changes to occur above the obstruction. (iii.) Cases in which the symptoms of acute obstruction supervene on those of a chronic stenosis. The anatomical changes are very similar to those in the preceding class, but some, such as distension and accumulation of fæcal matter above the obstruction, are emphasized. In discussing the morbid anatomy of intestinal obstruction, it will be convenient to describe, first, the changes found in chronic obstruction, then those of chronic cases which have terminated acutely, and, lastly, the lesions found in acute intestinal obstruction.

(i.) **The Morbid Anatomy of a Case of Chronic Obstruction.**—We are not concerned here with the causes of the chronic and progressive stenosis, which will be described below.

The intestine below the obstructed point is pale, contracted, and, as a rule, empty, and like the intestine of a starving animal. In a few cases, however, of stenosis in the small intestine, and especially when opium has been freely administered, a considerable bulk of fæces may collect in the colon. Thus, in a case of simple stricture of the small intestine under my care, the diagnosis was at first somewhat obscured by a large accumulation of hard fæces in the sigmoid flexure and rectum.

The changes in the segment of intestine above a stenosis are well marked and characteristic. They are distension, elongation, hypertrophy, inflammation, and ulceration of the mucous membrane, and in some cases striation of the mesentery from stretching. For a variable distance and degree the coils of intestine are dilated above every stenosis. But narrowing of the gut does not invariably obstruct the passage of the intestinal contents sufficiently to produce these changes. As the contents of the small intestine are habitually fluid or gaseous, a very considerable degree of narrowing may exist without offering any obstruction to the passage of the contents; while in the lower part of the colon, where the contents are solid, a much less degree of narrowing will produce pathological changes. In the early stages of a progressive stenosis, dilatation is confined to the coils immediately above the obstruction, and is only moderate in degree. When the lumen of the bowel has been nearly occluded, and especially when from any cause acute and total obstruction has supervened, the dilatation and distension may be enormous, and the whole of the intestinal canal above the stenosis may be effected. The contents of these dilated coils in the lesser degrees of distension are those normally found in the part of the alimentary canal involved. In the small intestine and in the upper part of the colon they will be fluid, but in the sigmoid flexure and the rectum large masses of solid fæces may accumulate above the obstruction ; but even in the lower colon in advanced degrees of stenosis the contents are, as a rule, liquid. As the obstruction becomes more complete, and these contents stagnate and ferment, an irritative secretion from the intestinal wall is added to them. As long as the circulation is not seriously interfered with, gas will not accumulate in the distended coils, for it is absorbed by the vessels of the intestinal wall. Moreover, gas always passes through the stenosis more readily than fluid fæces. The distended intestines necessarily increase in length ; this mechanical result of distension of an elastic tube is illustrated by the varicosity seen in veins and arteries submitted to excessive pressures. The greatly distended and lengthened intestines are thrown into folds, as is particularly well shown in volvulus of the sigmoid flexure, in which, on opening the abdomen, the gigantic coils of distended colon are found folded upon themselves and often occupying the whole of the abdomen.

Striation and Shortening of the Mesentery.—One of the functions of a mesentery is to provide a certain amount of slack peritoneum, so that, as the coil of intestine becomes distended, it may spread between the layers of the mesentery or mesocolon without hindrance. In the excessive distension above a stenosis, the mesocolon is often shortened by this means almost to the point of disappearance, and it may therefore be exceedingly difficult to bring a loop of distended sigmoid flexure out of a colotomy wound. In rare instances the whole of the slack peritoneum provided by the mesentery or mesocolon is utilized, and the strain thus thrown upon the peritoneum produces striæ in the line of the axis of the gut.

The factors leading to hypertrophy of the intestine above a stenosis are in part mechanical distension and in part irritation of the mucous membrane above the stenosis by the accumulated contents of the intestine. Patel asserts that hypertrophy does not occur in the absence of ulceration; in support of his contention that ulceration rather than obstruction is the determining factor, he points out that in intestinal obstruction due to pressure from without there is distension without hypertrophy, and, as is well known, that extensive ulceration without organic obstruction will produce well-marked hypertrophy. In the latter case, however, spasmodic stricture is probably present during life. Nothnagel investigated the nature of this hypertrophy in man, and his pupil Herczel by experiments on animals. The hypertrophy of the intestine is due to an increase in the muscular tissue and a dilatation of the smaller bloodvessels supplying the muscular coat. The connective tissue undergoes no change; the muscle fibres are not increased in number, but become larger, and more especially broader, the hypertrophy being manifest by the fifth day, and well marked by the ninth day, after the production of experimental stenosis. The degree and extent of the hypertrophy depend upon the amount of the stenosis and the time occupied in its production, hypertrophy being always best marked immediately above the stricture.

Inflammation and Ulceration of the Mucous Membrane above a Stenosis.—Enteritis nearly always exists above a fully developed stricture, and in most cases numerous large shallow ulcers are present. These stercoral ulcers present every transition from chronic indolent forms to sloughing and gangrenous patches. A group of such ulcers is found usually just above the stenosis, but others are scattered irregularly along the distended portion of the intestine. In obstruction of the colon there is often a group of characteristic ulcers in the cæcum, an event of importance, inasmuch as peritonitis frequently originates from these ulcers, either by the transudation of micro-organisms or by a gross perforation. These stercoral ulcers are seldom due to the irritation of hard impacted fæcal masses. They are the result of interference with the circulation, due to the distension combined with the chemical irritation of the intestinal contents and bacterial infection. Kocher has suggested that " distension ulcers " would be a better name.

(ii.) **The Morbid Anatomy of a Case of Intestinal Obstruction which has become Acute.**—The causes of such a sudden transition are numerous; for example, a foreign body, such as a plum-stone, may become impacted in a stricture, or a coil of distended intestine may be kinked or twisted, or the exhausted intestine may suddenly become paralyzed and relaxed. The morbid changes found in such a case will differ in degree only from those that have been already recounted. The distension and elongation will be far more extensive. The rapid accumulation of food, secretions, and gas, in the last few days of life, will distend such an alimentary canal from the stenosis to the stomach. The sudden rise of intra-abdominal tension will interfere with the circulation, and the gases will no longer be absorbed from the intestinal lumen, and will rapidly increase the distension, thus establishing a vicious circle. The shortness of the time will not permit of any increase of hypertrophy, but the enteritis and ulceration of the mucous membrane above the obstruction will be greatly increased, chiefly as a result of the sudden distension. Peritonitis from perforation or from the passage of micro-organisms through the coats of the intestine follows, frequently almost immediately after the obstruction becomes acute.

(iii.) **The Morbid Anatomy of a Case of Acute Obstruction of Normal Intestine.**—In nearly all forms of acute intestinal obstruction, not only is the lumen of normal intestine suddenly and completely interrupted, but a coil of intestine is strangled in addition. In some cases a band is knotted or noosed around it, in others the intestine passes through a hernial ring or an aperture in some membranous structure such as the mesentery or omentum; in some axial rotation takes place, and a volvulus is produced; and in others, again, a part of the intestine is invaginated within a part below and gripped by it. In whichever of these ways the acute obstruction is produced, three different conditions are shown by (*a*) the intestine below the obstruction, (*b*) the intestine above the obstruction, and (*c*) the strangled coil within the obstruction. An exception to this general rule is when a large gall-stone or foreign body passes suddenly into the normal intestine and abruptly interrupts its lumen. In these cases the first two conditions only of the intestine occur.

The changes found in acute intestinal obstruction are very different from those already described in a chronic stenosis. Hypertrophy and chronic inflammatory changes of the mucous membrane are entirely absent from the coils of intestine above the obstruction, but distension is, as a rule, even more marked in this portion of the gut. On the other hand, the changes in the strangled coil are profound and very evident, and the results of these changes on the general organism through the nervous and vascular systems produce the predominant symptoms of the disease.

(*a*) *The Morbid Anatomy of the Intestine below an Acute Obstruction.*—These coils are empty, contracted, and pale, and in the case of the small intestine no thicker than the little finger. No doubt at the moment of obstruction their normal contents are present, but when seen a day or two later at an operation or autopsy the coils are completely empty. This condition is probably brought about in the manner described in Dr. Bayliss and Professor Starling's experiments upon animals. The first effect of resisting the progress of intestinal contents, or of pinching or otherwise stimulating the intestine, is powerfully to augment the peristaltic waves above the stimulated point, and to paralyze and relax the intestine for 3 feet below it. In this way the intestine endeavours to overcome the obstruction and force on its contents. This augmentation of peristalsis is, however, evanescent, and soon the waves of peristalsis pass over the point of stimulation or obstruction and continue on along the intestine, which then becomes contracted and empty, and apparently passes into a condition of spasm. The condition of the intestine below a strangulation is therefore rather one of emptiness and spasm than of paralysis, as is so often stated.

(*b*) *The Morbid Anatomy of the Intestine above an Acute Obstruction of Normal Intestine.*—As has already been said, these coils of intestine do not present hypertrophy or ulceration. They are, however, greatly distended. Their walls are pale and thin when the obstruction is recent and very sudden ; when it is of longer duration, they are thickened by œdema and deeply congested with blood, but they are never the same dark purple colour as the strangled loop. Distension and dilatation are the most evident changes in the intestine above an acute obstruction. This distension may be local or diffuse. In many cases, and probably in all early cases, it is confined to the intestine immediately above the obstruction. In cases of some days' duration it is diffused, and the whole of the alimentary canal is dilated as high as the duodenum and the stomach. The contents of these dilated coils are for the most part liquid and pultaceous, and to only a small degree gaseous. Gas does not tend to accumulate until the circulation of blood through the intestines is so much interfered with that absorption ceases. This occurs, however, when general peritonitis complicates an acute obstruction. The degree of the distension of any coil of intestine is ultimately limited by the resistance of its own wall and the pressure exerted on it by the abdominal wall and adjacent coils of intestine. The generally accepted explanation of the origin of this distension is stasis of the intestinal contents. These contents consist not only of the food taken, which in such cases is very little, but also of the secretion of the various digestive glands. The secretion of saliva is, on an average, one or two pints a day, and each of the other glands probably supplies a similar amount. In acute intestinal obstruction with vomiting, probably a continuous and excessive secretion is produced by reflex irritation. The alimentary canal may roughly be divided into an upper part, consisting of the mouth, stomach, and duodenum, devoted to secretion, and a lower part, the colon, in which most of the water is abstracted from the intestinal contents. Nearly all forms of acute obstruction affect the small intestine, and in this way the area of secretion is shut off from the area of absorption. As a result the coils of intestine are flooded and distended by the secretion of the digestive glands, rendered still more excessive by the reflex irritation of the stagnating contents. The flood rises higher and higher from one coil of intestine to another until the duodenum and stomach are submerged, and it overflows in the gushing vomiting of the later stages of acute intestinal obstruction. The question of the fæcal character of this vomit is considered on p. 14. When the obstruction is in the colon, as in acute volvulus of the sigmoid flexure, the distension of the gut above the occlusion is much less marked, for here a part of the area of absorption is still available. It has been urged by some that a diffuse paralysis of the intestine, due to the lesion producing the obstruction, plays a part in the production of the dilatation of the intestine, and no doubt the toxic contents paralyze and relax the intestinal wall in the same way as nicotine and cocaine have been shown to do experimentally, but no amount of paralysis or relaxation would produce dilatation unless the contents accumulated or were increased by excessive secretion.

(*c*) *The Morbid Anatomy of the Strangled Coil of Intestine.*—A coil of intestine may be strangled in any of five ways: A bandlike structure may encircle its root and mesentery; it may pass through an orifice into a hernial sac, in which case the ringlike orifice acts in the same way as a band; a loop of gut may work its way through an aperture in the mesentery or omentum, the margins of the orifice producing strangulation; one portion of intestine may become invaginated into the coil below it, which tightly grips the intussuscepted portion and its mesentery, especially around its neck; or, lastly, a coil of intestine may, by axial rotation, twist the entering and leaving coils so tightly around the base of its mesentery as to obstruct the venous return and strangle the coil.

In whichever of these ways strangulation of a coil of intestine is produced, the results are the same. The pre-eminent consequences are vascular engorgement and violent stimulation of the sympathetic nerves supplying the coil. As a secondary result of interference with its circulation, the coil becomes distended to its utmost capacity with gas and with effused blood, and, lastly, the lumen of the alimentary canal is interrupted completely and suddenly by the mechanical pressure and by the paralysis of the strangled coil of intestine. The subject of strangulation of intestine has been investigated experimentally by Kader, whose results are summarized by Nothnagel. He placed ligatures of various tensions around coils of gut in animals, and observed the result. He also ligatured the mesenteric vessels separately and in combination with ligatures, occluding the gut at one point like an annular stricture.

The Vascular Changes in a Strangled Coil of Intestine.—When, for example, a band nooses a coil of intestine tightly, the veins returning from that coil are compressed, and become distended and engorged with venous blood. The arteries continue to force blood into the capillaries of the coil until they are filled, at a pressure approaching that of the arteries, and the whole coil is distended and erect with venous blood. Within an hour or so the coil becomes deep purple in colour, tense, œdematous, and exudes a blood-stained serum into its lumen and into the neighbouring peritoneum. This blood-stained fluid collects in the peritoneal cavity, or in the hernial sac, if one be present. In very rare instances the strangulation is so tight as to occlude the arteries at once; the veins are not distended with blood, and the coil of intestine dies and becomes gangrenous and flaccid in an hour or less. In such cases there is scarcely any distension of the strangulated coil. In other instances, which are still rare, the veins are occluded or nearly occluded at once, and the circulation ceases. In these cases distension, engorgement, and extravasation of blood occur, but the intestine dies in a few hours, the vessels become thrombosed, the arterial pressure is cut off from the coil, which exudes its serum and gas, and becomes flaccid, green, and gangrenous. In the vast majority of cases the obstruction of the veins is not complete, and enough blood escapes from the veins of the coil to enable a slow and greatly diminished circulation to be maintained, and for this reason such coils of intestine will often recover when released after three or four days of strangulation.

It cannot be too strongly urged that coils of gut are not dead and gangrenous because they are purple or black. The great majority will readily recovery when their veins are released. The prevalent notion that black gut is always gangrenous is responsible for many unnecessary enterectomies. True gangrene of intestine is shown by a green or grey colour combined with flaccidity and the presence of free gas and a stench in the peritoneum around the dead coil.

The Meteorism of a Strangulated Coil of Intestine.—When a coil of gut is moderately strangled, its lumen becomes distended to its utmost capacity with blood and with gas. This gas is probably carbonic acid, and is produced by the vital processes of the tissues. It is unable to escape as it should by the obstructed veins. The same phenomenon is always seen when intestinal clamps are applied to a coil of intestine in performing gastro-enterostomy or intestinal anastomosis. The coil of intestine is distended to many times its normal extent, and the tension so exerted draws into the constricting ring fresh coils of gut, which are in turn strangled and distended. These coils draw their mesentery along with them within the constriction, which in this way becomes tighter and tighter in a vicious circle. The extent and proportion of the abdominal distension and meteorism due to the strangled coil depend entirely upon its size and accessibility to palpation. Where it is small and inaccessible there will be no distension due to this cause, as when a mere knuckle of gut passes into a hernial sac or orifice or is strangled by a band. Where, however, a large coil of intestine is affected, as in volvulus of the sigmoid flexure, the abdominal distension which rapidly supervenes is almost entirely due to the strangled intestine, and may soon assume gigantic proportions. The tension of the strangled coils is, moreover, much greater than that of the coils distended by stasis above the obstruction. It is well to remember that strangled coils within the abdomen are as tense as an ordinary strangulated hernia.

The Infection of the Strangled Loop of Intestine and of the Peritoneum.—Even before the coil of gut is dead and gangrenous it loses its power of preventing the passage of micro-organisms from its lumen through its wall to the peritoneum, and as a consequence the tissues are infected and peritonitis is set up, which may spread to the whole peritoneum. Even if the strangulation be relieved at this stage, the patient frequently dies of this infection and the inflammation of the intestinal wall and peritoneum, which cannot be removed.

Thrombosis, Gangrene, and Perforation of the Strangled Intestine.—Ultimately the vessels become thrombosed and the gut gangrenous in all unrelieved cases. The gases and blood escape by exudation, and the strangulated coil is flaccid and green, whilst gas is found in the peritoneum and the exudate is stinking. In some cases crude perforations are present, but in many the orifices through which the gas and fluid contents of the bowel escape are microscopical. In a strangled coil of intestine, the capillaries, being unsupported and totally unprepared for the strain, rupture, hæmorrhage occurs into the substance of the bowel, and blood escapes into the neighbouring cavities.

A strangled coil of gut is paralyzed. This is probably a combined result of the distension, vascular interference, and the direct injury to nerves in the mesentery by the cause of strangulation. The lumen of the intestine is usually doubly occluded at the points where the intestine enters and leaves the constriction. Moreover, the high tension in the coil and its paralysis would form an effectual block to the passage of intestinal contents even when the entering coil is not mechanically closed, as sometimes happens in hernial orifices and apertures.

(B) **The General Consideration of the Symptoms of Intestinal Obstruction and their Pathological Explanation.**—The pathology of intestinal obstruction includes not only the morbid changes produced, but the resulting functional disturbances. Under the present heading these disturbances and their explanation will be discussed in a general manner.

Pain—(*a*) *Pain in Acute Intestinal Obstruction.*—Three varieties of pain are found when the intestine is strangled. The first pain is usually sudden in its onset and intense, doubling the patient up in his agony. It originates in the strangled coil, and is due to the mechanical injury of the constriction and to the stretching and distension of the coil by the venous engorgement and the gaseous accumulation. It is as a rule referred to the umbilicus in strangulation of the small intestines. This is a special instance of the general rule that sensations can only be localized in the segments of the alimentary canal, which are more or less fixed —namely, the stomach, duodenum, and colon. Sensations from the small intestines, which wander freely about the abdomen, are experienced as diffuse pains, and are referred to the umbilicus. Thus, as I have pointed out, when a large gall-stone passes into the duodenum, the pain is at first correctly localized to the epigastrium, but when the calculus has reached the small intestine the pain is diffuse, and is referred to the umbilicus. Similarly, in volvulus of the sigmoid flexure or of the ileo-cæcal junction the pain is, as a rule, correctly localized in the iliac fossæ, and the same applies to intussusception or impaction of a foreign body at the ileo-cæcal junction. In rare instances the pain has been definitely localized in very various parts of the abdomen, although the lesion was in the small intestine, but for these cases no explanation has been offered. This first pain is continuous, so that the patient has not even momentary relief, until after a time the nerves of the strangled coil become exhausted and poisoned, and the pain abates. It disappears entirely just before death, and in some of these cases gangrene of the intestine has been found; in others a perforation has allowed the coil to collapse, or the remission of pain must be ascribed to the narcosis of septicæmia and profound collapse. In exceptional cases the pain has been insignificant from start to finish. Some of these patients have been very old; in others the strangulation does not appear to have been abrupt in onset. This pain originates in the sympathetic nerves, and is characterized, as such pains usually are, by the association with vomiting, collapse, and profound vascular disturbance. It is more or less proportional to the extent of intestine implicated in the strangulation, and to the severity of the constriction. The higher up in the intestine the lesion is situated, the more severe as a rule is the pain; strangulation of the small intestine is always more painful than that of the colon, probably because the vagi take a larger share in the formation of the solar and mesenteric than in that of the hypogastric and inferior mesenteric plexuses. This explains why strangulation by bands and internal hernias, which as a rule implicate the small intestine, are much more painful than volvulus, which is usually found in the colon.

The second pain is of the nature of colic. It is due to the tetanic and violent peristaltic efforts of the intestine above the obstruction to force the contents past the obstacle. These paroxysms of colic do not last very long, and become less and less severe and frequent until they stop. Their cessation is accounted for partly by the rule that no hollow muscular organ, whether intestine, bladder, or uterus, will struggle long with an insuperable obstacle; partly by the distension which thins out the muscular wall and places it at a great mechanical disadvantage; and partly by the poisoning of the neuro-muscular apparatus by the toxins formed in the stagnant contents. As a consequence of the last two causes the gut is frequently paralyzed in long-standing cases, and is unable to empty itself even when the obstruction is relieved.

The third pain is stabbing, localized, and associated with tenderness and rigidity, and often with some rise of temperature. Unlike the first and second pains, it is not relieved but is increased by pressure, which, indeed, cannot be borne. It is due to the onset of peritonitis and infection, and appears from the third to the fifth day, when micro-organisms begin to pass from the lumen of the intestine through the wall to the peritoneum.

(*b*) *In Chronic Intestinal Obstruction.*—The pain is usually of the peristaltic variety only. The lesion producing the obstruction is, as a rule, almost painless, and there is no intestinal strangulation. Peritonitis frequently appears at the end of cases of stenosis, but it is far from constant, and is to be regarded as a complication. The onset of pain in this form of obstruction is as a rule gradual. At first it only gives rise to periods of discomfort associated with the rumbling of wind which "will not pass downwards." Gradually, as the stenosis becomes more complete and the coils of intestine above it more distended and hypertrophied, definite attacks of colic occur, and these become more frequent and severe. In these attacks the patient often cries out, is doubled up with the pain, often grasps his abdomen, and endeavours to rub away the lump formed by the tetanically contracted intestine. In other instances handling the abdomen appears to excite attacks, and is avoided by the patient. In obstruction of the colon the pain is often localized correctly by the patient, who is able to trace the peristaltic wave along the course of the large intestine with ever-increasing tension and distension to a climax of tetanic spasm at the stricture. The intestine can often be seen to rise up as a writhing, rigid mass at the height of the spasm.

Vomiting—(*a*) *Vomiting in acute intestinal obstruction* commences from half an hour to twenty-four hours after the onset of the pain, usually after a short interval. To some extent its onset and degree are determined by the severity of the pain, the presence or absence of food in the stomach, and the sensibility of the patient. Some people, especially children, vomit readily, others with great difficulty or hardly at all. The early vomiting of acute intestinal obstruction is associated with intense nausea, straining, and eructations, and gives no relief to the patient's sufferings. At first the vomited matter consists of gastric contents, but as time goes on it becomes green and bilious. The violence of the vomiting may to some extent lessen after a day or two. Finally the character of the vomiting alters, and it becomes gushing; the patient at frequent intervals opens his mouth, from the corner of which, without effort or straining, pours a foul liquid stream. The vomit has, as a rule, at this stage become fæcal; it is entirely liquid, brown in colour, and of a horrible odour, more or less resembling that of fæces. It greatly exceeds in quantity any fluids which may have been taken. It is at this stage that "drowning from fæcal vomiting" is apt to occur when an anæsthetic is administered. As the patient becomes anæsthetized the sensibility and contractility of the pharynx, larynx, and œsophagus, are annulled; suddenly the patient stops breathing, and a filthy liquid stream commences to pour in overwhelming quantity from his nose and mouth. Unless the head and neck be immediately lowered and turned upon the side, and the mouth forced open and the lower cheek depressed by a finger passed into it, the patient inhales the foul fluid into his lungs, and either dies at once, or, if his lungs are rapidly cleared, a day or two later, of septic broncho-pneumonia.

The early vomiting of acute strangulation is a reflex phenomenon of the sympathetic nervous system, and resembles that seen in biliary and renal colic, in crushing of a testis, in torsion of a testis or ovary, or when the peritoneum is violently stimulated by an acute infection or injury. The regurgitation of bile into the stomach during the second stage of vomiting has not been satisfactorily explained. It would appear that there is a greatly increased flow of bile into the duodenum; and if this be admitted, then, as Pawlow has shown, the pyloric sphincter would be relaxed by the alkalinity of the bile. Even then it is hard to see why it enters the stomach in any quantity, unless antiperistalsis exists in the upper part of the alimentary canal. John Hunter, in his paper on Intussusception, maintained that antiperistalsis occurred during vomiting, and for this reason recommended emetics as a desperate means of undoing intussusceptions.

Gushing and fæcal vomiting are due to the stasis and acumulation of intestinal contents above the obstruction. The profuse secretion of the salivary, gastric, biliary, pancreatic, and intestinal glands floods the intestines above the occlusion, distends the stomach, often to an extreme degree, and finally overflows from the mouth. This explanation of gushing fæcal vomiting was, according to Leichtenstern, first propounded by Huguenot of Montpellier (1713). It was later advocated by van Swieten and Henle. In support of this explanation, it may be pointed out that the degree and early onset of gushing vomiting are practically proportional to the proximity of the intestinal occlusion to the stomach. When the obstruction is low down in the colon, not only is a much longer portion of the intestine available to contain the secretion, but the major part of the "area of absorption" is above the obstruction, and the water can be abstracted from the intestinal contents in a normal manner. Thus, in gallstone intestinal obstruction, in which the obstruction is at first in the duodenum, the vomiting at the very start is gushing and copious, but abates as the calculus moves farther and farther down the canal. On the other hand, in volvulus of the sigmoid flexure gushing vomiting is often entirely absent.

The fundamental error, which has vitiated the whole problem of fæcal vomiting, and has produced a superabundant literature, is that the foul matter smelling like fæces must have been produced in the lower segments of the intestine, and have been conveyed backwards to the stomach. At first it was supposed that this was true fæcal matter, and that its presence proved that the obstruction was in the colon. This error was long ago exposed by the observations of morbid anatomy; but modern writers, such as Nothnagel, still think it necessary to explain the transportation of intestinal contents from a position immediately above the occlusion to the stomach. There does not, however, appear to be any evidence that this transference occurs, and it is therefore needless to explain it. It sometimes happens that innumerable fruit-stones accumulate above an intestinal stricture, and many such cases have terminated acutely with fæcal vomiting; but the stones have always been found massed in the ampulla above the stricture, and not distributed up the intestine. The same remark applies to metallic mercury, which in former times was given to overcome the obstruction.

The chief hypotheses to account for the regurgitation of intestinal contents from the lower segments of the intestine to the stomach may, however, be stated briefly. The oldest and most persistent explanation—that of antiperistalsis (motus antiperistalticus)—originally advanced by Galen, was supported by John Hunter, and in modern times by Nothnagel, although with reservations. The experimental researches of Dr. Bayliss and Professor Starling have shown that, when Auerbach's and Meissner's plexuses are paralyzed, short rapid waves pass indifferently up or down the intestine. These waves are purely muscular in origin, and have very slight propulsive force. True peristalsis is a neuro-muscular act, and requires for its production the integrity of the whole muscular and nervous apparatus of the intestine. Dr. Bayliss and Professor Starling do not state that they ever succeeded in reversing true peristalsis in their experiments; but W. B. Cannon, by incorporating bismuth subnitrate in the food and enemas given to cats, was able to observe by X rays and the fluorescent screen that the commonest movement of the ascending and transverse colon was one of antiperistalsis in this animal; large enemas were even driven through the ileo-cæcal valve into the lower coils of the ileum. According to Nothnagel, a few indubitable cases have been recorded of the vomiting of formed fæces even before the physician's eyes, as in the cases narrated by Rosenheim, Desnos, and Schloffer. Some years ago a hysterical woman, under the care of Sir F. Treves, not only vomited true fæcal masses, but enemas containing castor-oil, and subsequently methylene blue. Laparotomy was performed on this woman on no less than three occasions by different surgeons to cure a gastro-colic fistula, which was, however, shown to be absent at each operation. Antiperistalsis undoubtedly exists in very rare cases, but, as has already been pointed out, it is not necessary to invoke it in the explanation of fæcal vomiting.

Brinton advanced the hypothesis of a regurgitant axial current. He asserted that the ordinary waves of peristalsis in an occluded coil of intestine produce a forward movement of the contents along the sides, and a reversed current up the centre of the gut. It has been proved, however, that the distended intestine is soon exhausted, and to all intents and purposes immobile as regards contractions. Others have suggested that the pressure of the diaphragm and abdominal muscles, especially during the act of vomiting, would empty the distended lower coils into the empty coils and stomach above them. It has been urged that, since the coils cannot be emptied in a downward direction, the contents must be forced upwards. This explanation, though plausible, is for mechanical reasons unsound ; for in the abdominal cavity the upper coils would be submitted to the same rise of pressure as the distended ones, and no transference of contents would take place ; to bring about this result the pressure would have to be concentrated on the distended coils.

Fæcal vomit is of a brownish-yellow, brownish-green, or yellowish-grey colour, and has an offensive odour comparable to that of fæces. No chemical analysis of fæcal vomit has apparently ever been made, but the colour is probably due to bile-pigments reduced to urobilin by the decomposition going on in the fluid. In part it may be due to altered blood-pigment exuded from the congested and inflamed mucous membranes. The fæcal odour is due to the products of the bacterial decomposition of proteins, which requires considerable time, and in normal circumstances is not complete until the contents of the bowel have reached the lower part of the colon. Should the intestinal canal be obstructed so that the contents remain in the duodenum and stomach for a day or two, this decomposition is then completed there, and a fæcal odour is produced; nor is there any need to invoke antiperistalsis or a regurgitant axial stream to transport the decomposing matter from just above the point obstructed.

(*b*) *Vomiting in chronic intestinal obstruction* is not so urgent, persistent, or severe, as in strangulation of the intestine. As a rule it occurs at intervals, and then after food only. The patient as a rule discovers that, so long as he is strictly moderate in the quantity of food taken, and for the most part restricts himself to liquids, he can control the vomiting. Sometimes vomiting occurs during severe attacks of colic. It is then comparable to the vomiting seen in any other form of colic, and characteristic of all sympathetic pains. In several cases of cancerous stricture of the lower part of the large intestine, with great distension of the transverse colon, it has appeared to me that the copious vomiting which occurred at intervals would bear the following explanation: The commencement of the transverse colon crosses the second part of the duodenum at right angles without the interposition of peritoneum or any mesentery, and when greatly distended must press upon the duodenum at this point, and more or less obstruct it. The stomach was distended in these cases, and it appeared probable that this was due to a secondary mechanical obstruction produced in this way, and without the intervening small intestine having as yet become fully dilated. This explanation would accord with the intermittent and irregular character of the vomiting, and with the observation that it is sometimes very large in quantity, as in pyloric obstruction, and is always induced by a full meal.

Collapse.—In acute intestinal obstruction collapse is one of the earliest manifestations, but in the chronic form it does not occur until the final and closing stage, when the obstruction has become acute. In collapse the patient is prostrated with weakness, he is anxious and restless, the pulse is rapid, small, and thready, the temperature subnormal, and the skin and extremities cold and clammy with sweat.

Collapse in acute intestinal obstruction is the result of three causes, any one of which can produce collapse, but in acute intestinal obstruction they are combined until the profound and terrible collapse which immediately precedes death results. These causes are—(1) An acute lesion of the terminations of the splanchnic and pneumogastric nerves in the abdomen; (2) depletion of the fluids of the body by the profuse vomiting and sweating, which produce a concentration of the blood; (3) intoxication and infection by the decomposing products, arrested by the obstruction, and by bacterial invasion. All three of these causes play a part at various stages in the production of collapse in acute strangulation of the intestine. In the terminal collapse of chronic obstruction the third is the predominant cause and to a less degree the second, whilst the first takes scarcely any part. (1) The reflex nervous factor has been fully investigated experimentally by Bezold, Goltz, Bernstein, and Ludwig and Asp. The first effect of a violent stimulation of the vagus in the abdomen is to slow the pulse, and this is sometimes seen in the early stages of any acute abdominal lesion. The subsequent result is paralysis of the cardio-inhibitory centre, and the pulse becomes rapid. Severe lesions of the vaso-constrictor branches of the splanchnic nerves at first produce a transient constriction of the vessels and a rise of blood-pressure, which in turn slows the rate of the heart. This rise soon passes off, and the abdominal arterioles are paralyzed and the viscera become engorged with blood, the blood-pressure falls, and the heart beats faster. The superficial vessels are depleted of blood, the radial and other accessible arteries are small, and the pulse thin, thready, and weak. The skin is pale and cold, especially in the extremities — the hands, feet, nose, and ears. The cerebral circulation is small and precarious, and to this must be attributed the anxiety, fear, and restlessness. The cerebral anæmia also reduces the tone of the cardio-inhibitory centre, and in this way quickens the pulse. The centre which controls sweating is also interfered with, and the patient is bathed in a cold, profuse perspiration. Probably the thermogenic centres are also more or less paralyzed, for, as Leichtenstern has pointed out, not only are surface temperatures depressed, but those taken in the rectum. (2) The depletion of water by vomiting and perspiration. In acute intestinal obstruction the patient frequently vomits pints of fluid in addition to that which he loses by profuse sweating and passes as urine. Any fluid drunk is often rejected at once by the stomach, and the attempt to take liquids by the mouth is abandoned. As a result there is rapid loss of weight, the blood is concentrated and becomes more viscous, and probably all the tissues of the body are similarly depleted. The most obvious sign of this rapid depletion of fluids is the falling-in of the cheeks and eyes. The latter become "ringed" and the whole face is pinched. The tongue is dry, small, and wrinkled, the secretion of saliva stops, the skin is wrinkled, and the urinary excretion is diminished or even suppressed. Whilst this is mainly due to the shortage of fluids in the body, the great fall of blood-pressure no doubt also plays a part, especially in the failure of the glomerular excretion of the kidney. At one time it was held erroneously that the diminution of urine was due to only a smaller part of the intestine being available for absorption, and therefore that the degree of anuria was an indication as to how high up in the alimentary canal the obstruction was placed. This is true only in so far that vomiting and collapse are proportional to the proximity of the obstruction to the stomach, and it is these factors which diminish the excretion of urine. According to Nothnagel, the concentration of the blood may reach 24 per cent. of its volume, and the percentage of hæmoglobin is correspondingly increased. This is no doubt an important factor in reducing still further an already weakened circulation, and, as in severe cholera, is probably responsible for muscular cramps, especially in the calves, and for such unusual and rare cerebral symptoms in intestinal obstruction as coma, delirium, and convulsions. (3) Intoxication and infection from the obstructed bowel. In the final phase of acute intestinal obstruction or of chronic obstruction which has become acute and absolute, the patient presents a picture of profound poisoning. He lies prostrate and collapsed in bed, wasted to a grey gaunt shadow, with sunken eyes and fingers wandering aimlessly over the bedclothes. The skin is covered with a clammy sweat, and his parched, cracked tongue can produce nothing but a hoarse whisper. His pulse is uncountable, and his breathing irregular, sighing and flickering as though about to cease for ever. Yet he seems to suffer little pain, only to be haunted by a nameless dread. This final stage of collapse is due to the absorption of poisons from the obstructed coils, and less frequently to septicæmia. The list of poisons is extensive, but it is uncertain which, if any, produce these symptoms of sapræmia. These poisons are the products of the decomposition of proteins in the stagnant contents. Indol, phenol, skatol, and aromatic oxy-acids, hydrogen disulphide, acetone, and diacetic acid, have all been credited with a share in the production of this condition. Others have regarded the poison as an alkaloid, or the product of the bacillus of malignant œdema or of the colon bacillus. Some special symptoms may be attributed to this terminal sapræmia and septicæmia, such as delirium, coma, pyrexia not due to peritonitis, and nephritis when it occurs.

In conclusion, it may be pointed out that collapse due to reflex nervous causes is abolished in an hour or so by morphine, and that that due to depletion of fluids can be rapidly met by saline transfusions, so that these two causes together need offer no insurmountable obstacle to recovery after the cause of the obstruction has been removed. On the other hand, when the collapse of obstruction is due to sapræmia or septicæmia, the most successful operation will usually fail to save the patient.

The Condition of the Bowels in Intestinal Obstruction.—In partial obstruction a diminished quantity of fæces and flatus passes through to the intestine below, and the fæces frequently accumulate for some time in the colon before they excite the desire to defecate. The flatus is voided daily, and an enema will at any time readily empty the colon of the fæces and flatus it may contain. This condition is known as one of "irregular and partial constipation." When the mucous membrane of the coils above the stenosis is inflamed and ulcerated, frequent fluid motions may be passed containing blood and mucus. This condition is one of "irritative diarrhœa." When acute strangulation has taken place, the bowels are as a rule in a condition of "absolute constipation." Neither fæces nor flatus are voided, nor has the patient any desire to defecate. If an enema be administered to such a person, it is often retained, or is returned with little force and without result in flatus or fæces, except, perhaps, such as may be washed mechanically from the rectum. A motion is sometimes passed immediately after the onset of the pain and vomiting. On the other hand, one may be voided just before death and after days of absolute constipation. The condition of the bowel below a strangulation appears to be a most curious one. It has been customary to speak of it as paralyzed, but when seen at operations or autopsies it is empty, contracted, and pale, and not relaxed and flaccid. The condition is rather one of inhibitory spasm. When a motion is passed soon after the onset of the attack, it is probably in consequence of this spasm being established. When one is passed just before death, it is because asphyxia has stimulated peristalis. In some cases the voided matter is supposed to have come from above the obstruction either by means of a perforation or owing to a relaxation of the parts at the moment of death. In acute strangulation of the colon tenesmus is frequently present. Thus, in intussusception situated at the ileo-cæcal valve, frequent small motions of blood and slime are passed. Tenesmus and straining are not uncommon in volvulus of the sigmoid flexure or in acute obstruction of a cancerous stricture of the colon. In very rare instances a strangulated hernia has been associated with profuse diarrhœa ("choléra herniaire," Malgaigne). Diarrhœa is even rarer in internal strangulation. The fluid is supposed to be a profuse secretion from below the obstruction, due to paralysis of the mesenteric nerves. Some of these cases have so closely resembled cholera as to be mistaken for it.

Indicanuria.—Indol is produced chiefly in the small intestine by the bacterial decomposition of proteins. It is excreted in the urine as indican (potassium indoxyl-sulphate). In acute obstruction of the small intestine its amount may rise to 100-150 milligrammes daily (Jaffé), the normal quantity being from 5 to 20 milligrammes. This increase may be used in the differential diagnosis between obstruction in the small intestine and in the colon. When indicanuria is well marked on the second or third day of an obstruction, the occlusion is in the small intestine.

When it is absent after several days of subacute obstruction, the lesion is in the large intestine. Its presence in such a case, however, does not exclude a lesion of the large intestine, for the distension may have spread into the small intestine. Indicanuria also occurs in peritonitis, especially when diffuse, in tuberculous and typhoid ulceration of the small intestine, and in simple enteritis and cholera. It is also found in the chronic decomposition of pus in cavities, especially the pleural cavity.

3. The General Symptoms of Intestinal Obstruction.

Its Three Clinical Varieties.—The three clinical varieties of intestinal obstruction are—(*a*) *Acute intestinal obstruction;* (*b*) *chronic intestinal obstruction;* (*c*) *cases of chronic intestinal obstruction which have terminated acutely.* These three forms of intestinal obstruction present the same symptoms, but in a very different degree and variety. The classical symptoms of intestinal obstruction in their usual order of incidence are, pain, vomiting, collapse, constipation, and the various changes which may be observed on examining the abdomen. The following classification summarizes the differences presented by these symptoms in the three clinical varieties:

	Pain.	Vomiting.	Collapse.	Constipation.	Abdomen.
(*a*) Acute intestinal obstruction.	Sudden, severe, continuous, stabbing.	In half an hour; severe, continuous, gushing. fæcal.	Early and profound; temperature subnormal; cramps, thirst.	Absolute, and to enemas.	Usually normal at first.
(*b*) Chronic intestinal obstruction.	Gradual, intermittent, colicky with flatulence.	Late; only after food, seldom fæcal.	Late, slight; temperature normal.	Partial, alternating with diarrhœa, blood, and slime; morning diarrhœa.	Distended, visible contracting coils of gut.
(*c*) Chronic obstruction become acute.	1. The symptoms of (*a*) supervene on a history of (*b*). 2. Onset less sudden and severe than in (*a*). 3. Abdomen distended and visible contracting coils of gut. 4. History of previous attacks cured by enemas and purges.				

Pain is the earliest symptom in all forms of intestinal obstruction. In acute intestinal obstruction, the patient, who may be in perfect health, is suddenly struck down by the most severe abdominal pain without any warning. The pain is so exceedingly severe that it doubles him up, is continuous, and never remits. At first it is stabbing, and is due to the actual damage to nerve-endings in the intestine and peritoneum. The second pain comes on in exacerbations of the first, and is of a colicky character. The third variety of pain does not supervene until the strangled coil has become inflamed and peritonitis has commenced, and it is therefore associated with tenderness on palpation. The pain in chronic intestinal obstruction forms a marked contrast to the above. The patient has been in ill-health for some considerable time, suffering from flatulence, dyspepsia, and more or less abdominal unrest, and he has perhaps wasted. The pain gradually becomes worse during twenty-four or thirty-six hours, until the patient has severe attacks of colic; but these occur only at intervals, and between the attacks he is free from pain. These attacks of pain are due to the peristaltic and tetanic contractions of the hypertrophied intestine above the stenosis.

Vomiting in acute intestinal obstruction usually comes on half an hour after the pain, but it may be delayed for twelve hours. It is characterized by nausea and severe retching, and continues without intermission, becoming worse and worse until it is fæcal in character, from the second to the seventh day. The vomit at first consists of gastric contents, then becomes bilious, and finally is a fluid fæcal stream of gushing vomit, which nothing will arrest except a large dose of opium. In chronic intestinal obstruction the vomiting does not occur until the third or fourth day, and is never a prominent symptom of the disease. As long as the patient abstains from food and is quiet, he does not vomit, although he may feel sick. Gushing fæcal vomiting is seldom seen, and then only when the case has terminated acutely. The contrast between the vomiting in acute and in chronic intestinal obstruction may be summed up as follows: The higher up in the alimentary canal the cause of the obstruction is, the more severe, profuse, and continuous, will the vomiting be, and the less the distension. On the other hand, the lower the obstruction is in the intestinal tract, the greater will be the distension, and the less and later will the vomiting be. Acute obstruction as a rule implicates the small intestine; chronic obstruction, usually the colon.

Collapse is the third symptom to appear in acute intestinal obstruction, and it is usually well marked in a few hours. The patient is pale, cold, and sweating. The hands and feet and the tips of the nose and ears are cold and clammy. The pulse is a feeble thread, or perhaps impalpable. At first it may be slow, but soon its rate is increased. The temperature is subnormal. The patient suffers from prostrating weakness, dreadful thirst, and often from severe cramps in the calves of the legs. The final toxic stage of collapse has been described on p. 19. In chronic intestinal obstruction, on the contrary, the collapse is not often severe until late in the disease, when the patient is distended and poisoned by the products of the bacterial decomposition in his alimentary canal. The temperature is as a rule normal or slightly raised.

Constipation in acute intestinal obstruction is as a rule absolute. It is true that the bowel may empty itself immediately after the onset of the pain and vomiting, but after that neither fæces nor flatus are passed, nor has the patient any desire to defecate. The best diagnostic method of testing for the presence of absolute constipation is the double turpentine enema. Two enemas, containing half an ounce or an ounce of turpentine in half a pint of soap and water, should be given with an hour's interval. The first enema is intended to wash away such fæcal matter as may be contained in the rectum. The second is, however, the enema upon which attention should be concentrated, and three points should be carefully noted concerning its return : first, the amount of fæcal matter and bile evacuated ; secondly, whether flatus is returned with and after the enema (and for this reason care must be exercised that air is not injected with the enema) ; thirdly, whether the enema is returned with force, or merely flows away, or is retained, for this is a gauge of the degree of inhibition of the colon. In acute intestinal obstruction the second turpentine enema is retained or flows back without much force, and brings away neither fæces nor flatus. The exceptions to this general rule are the tenesmus which occurs in intussusception, and sometimes in volvulus, of the sigmoid flexure, and the very rare form of diarrhœa sometimes associated with any form of strangulation.

The constipation in chronic intestinal obstruction presents several curious characters. It is incomplete and irregular, the patient passes flatus at all times and fæces at irregular intervals, and a turpentine enema will always readily empty the rectum of its contents of flatus and fæces. Frequently diarrhœa alternates with constipation, or scybala are passed in a fluid stool. Sometimes there is a passage of blood mixed with slime. One of the earliest signs of an ulcerating cancerous growth low down in the colon is "morning diarrhœa"—that is to say, as soon as the patient rises in the morning he is obliged to pass a fluid motion. If this sign appear in a man over forty, who has had no previous abdominal trouble, and especially if it be associated with flatulence, the rectum should be examined, and the abdomen palpated for a carcinoma of the colon.

The Abdominal Examination in the early stages of acute intestinal obstruction as a rule reveals very little. The abdomen is usually flat, not distended, rigid, or tender. In intussusception and volvulus there are, however, well-marked signs. An intussusception can be felt under an anæsthetic in about three-quarters of the cases as a sausage-shaped, tender tumour, which hardens and softens. A volvulus will greatly distend the abdomen, and render it extremely tender quite early in the disease.

In chronic intestinal obstruction the abdomen is distended, frequently to an excessive degree. The most certain sign, however, of chronic obstruction is visible peristalsis. When in a case of stenosis of the intestine the light is arranged so as to strike transversely across the abdomen, and the latter is gently kneaded, coils of intestine will very frequently be seen to rise up and harden in vermicular contractions, and waves of peristalsis will be seen to pass from one part of the abdomen to another. These movements are usually associated with borborygmi. The condition of intestine revealed by this sign can only have been produced by a partial intestinal obstruction of some one or two months' duration. This obstruction has produced hypertrophy and distension of the coils of gut above the stenosis, and at the same time emaciation has thinned the abdominal wall. These three factors combine to render the waves of peristalsis visible. In extreme emaciation the normal intestine may be seen in a state of peristalsis through the abdominal wall, but the coils do not harden as they contract.

Cases of Chronic Intestinal Obstruction which have terminated acutely.—In the third clinical variety of intestinal obstruction, the symptoms of acute obstruction supervene on a history of chronic obstruction. The history is usually somewhat as follows: The patient is as a rule over forty years of age, and has enjoyed good health until some six or nine months ago, when his health began to fail; he became flatulent, dyspeptic, troubled with abdominal unrest, and began to lose flesh. Gradually attacks of colic came on, and became more frequent and severe as time went on. These attacks were accompanied by severe pain, abdominal distension, constipation, and perhaps by vomiting. These attacks were, however, relieved by vigorous purgatives and enemas. The present attack commenced like the others, with a more or less gradual onset, and after some twelve hours or so reached a climax. When seen, the patient presents all the symptoms of acute intestinal obstruction, but in a less severe form than in strangulation; the pain is less terrible, the vomiting not so persistent and severe, and collapse not well marked. His bowels, on the other hand, are absolutely constipated, and enemas produce no result. Moreover, in marked contrast to nearly all cases of strangulation of the intestine, his abdomen is distended, and shows coils of intestine in vigorous peristalsis. This later sign clearly indicates that, however acute the present attack may be, progressive obstruction has existed for some months.

Opium.—The symptoms characteristic of the three forms of intestinal obstruction are clear and obvious, but they can all be made to disappear by the administration of opium in large doses without changing the gravity of the condition or preventing the progress of the case to a fatal termination. On *a priori* grounds opium should never be administered to an acute abdominal case, until either the diagnosis has been established or the patient has finally refused operation. The result of the administration of a large dose of opium to a case of acute intestinal obstruction is surprising. The pain is relieved within an hour, and the vomiting ceases; the patient grows warm and comfortable, the collapse disappears, and the pulse becomes once more full, regular, and strong.

It is often essential to know whether opium has been already administered to an acute abdominal case, for if so allowance must be made in the consideration of every sign and symptom, and little weight attached to their absence in such a case. The signs which indicate the administration of opium are the pin-point pupil, the purple flushed cheek, and a dreamy tone of voice, easily recognized with a little practice.

4. THE DIAGNOSIS OF INTESTINAL OBSTRUCTION.

The diagnosis of a case of intestinal obstruction may be divided into three stages: (i.) The general diagnosis. A careful review of the signs and symptoms of the case will show whether there is mechanical obstruction of the intestine. (ii.) The differential diagnosis of the different causes of intestinal obstruction. When it has been proved that the bowel is obstructed, and by some organic, and for the most part remediable, cause within the abdominal cavity, the next step is to ascertain the nature of the cause. (iii.) The differential diagnosis from similar conditions. The passage of food along the intestine may be arrested, however, by many conditions which are not classed under organic intestinal obstruction, and in the third stage of diagnosis these dynamic conditions must be excluded. Such are external hernia, peritonitis, pancreatitis, embolism and thrombosis of the mesenteric vessels, toxic conditions, and nervous derangements of the intestines.

(i.) **The General Diagnosis of Intestinal Obstruction.**—The diagnosis of intestinal obstruction is made from the presence of the symptoms considered in the last section. A careful review of the case will not only show that organic intestinal obstruction is present, but will also serve to place the case under its appropriate clinical heading of acute, chronic, or a chronic case which has terminated acutely. The common causes of each of these varieties may now be enumerated.

(A) *Acute intestinal obstruction* is usually due to intussusception, bands, volvulus, gall-stones, internal hernia, or apertures. (B) *Chronic intestinal obstruction* is usually due to cancerous stricture of the colon, fæcal accumulation, chronic intussusception, stricture of the small intestine, pressure on the intestine from without. (C) In *cases of chronic intestinal obstruction which have terminated acutely*, the causes of the chronic obstruction are those given above. The acute termination is due to the impaction of a foreign body, bending, kinking, or volvulus of the intestine above the stenosis, or to acute paralysis of the intestine. This classification is convenient, and for the most part trustworthy; but all the causes of acute obstruction given above may exceptionally produce subacute or even chronic symptoms, and they are then very liable to be wrongly diagnosed.

Up to this point the diagnosis of intestinal obstruction is comparatively easy, but the solution of the problem as to the exact cause responsible for the obstruction requires an investigation conducted with the most critical care, and it must be admitted that it is often impossible to come to a reliable conclusion.

(ii.) **The Differential Diagnosis of the Different Varieties of Intestinal Obstruction.**—In order to discover which particular form of intestinal obstruction is present, two methods may be adopted. The investigator may compare the descriptions of a complete list of causes with the case in point, and select that which most nearly tallies. This method of diagnosis is only possible for a medical man who from long experience is able to recognize diseases, as he does faces, at sight; others must be content to review carefully each point in the history, symptoms, and signs, in turn, and thus determine to which particular cause it points. A review of the information so gained will frequently lead to an almost certain diagnosis.

A *history* of previous localized peritonitis will suggest that the obstruction may be due to a peritoneal or omental band, or to adhesions, kinks, or bends. Under this heading come appendicitis, pelvic peritonitis, cholecystitis, and tuberculous peritonitis. External hernias which have been strangulated and then reduced are a prolific cause of intestinal obstruction by omental and peritoneal bands, whilst in other cases a hernia has been reduced *en masse* within the abdomen just before the onset of the symptoms of obstruction. A history of long-continued pain in the region of the liver, and perhaps of jaundice and colic, can be obtained in practically every case of gall-stone intestinal obstruction (*vide* p. 85). Definite symptoms of cancerous stricture of the colon are usually preceded by some six months of failing health, in a middle-aged patient, associated with abdominal unrest, dyspepsia, flatulence, irregularity of the bowels, and wasting. In other cases a definite history may be obtained of dysenteric or tuberculous ulceration of the intestine, which may indicate the probable cause of a stenosis. In very rare instances a history may be obtained of a separated intussusception or the passage of an enterolith.

Symptoms—Onset and Duration.—As the case can readily be classed as acute or chronic from the character of the onset and the symptoms, the causes of acute and of chronic intestinal obstruction given on p. 26 must then be recalled. When the *pain* is sudden in onset and continuous without any period of relief, the case is one of acute intestinal obstruction, and the cause is one of those detailed above. On the other hand, when the pain is colicky and intermits, it is due to recurrent waves of peristalsis, and the obstruction is chronic and partial. The same holds good with regard to the degree and rapidity of onset of the *collapse.*

When collapse is well marked within twenty-four hours, some form of strangulation is as a rule indicated. As regards the position of the obstruction, it may be stated generally that the more frequent, profuse, and urgent the *vomiting*, the higher up in the alimentary canal is the obstruction; whilst when vomiting is infrequent, or only occurs after food, it may be inferred that the obstruction is low down in the alimentary canal. The highest cause of obstruction is the passage of a large gall-stone from the gall-bladder directly into the duodenum, and the vomiting in these circumstances can only be described as tremendous, and it is often said to be fæcal from the start; but as the stone passes down the intestine the vomiting becomes less severe. Bands, internal hernias, and apertures, implicate the jejunum and ileum, and in these conditions the vomiting is frequent, severe, early becomes fæcal, and is comparable to the vomiting seen in a strangulated external hernia. Intussusception is nearly always in the ileo-cæcal region, and vomiting is not a special feature. The child usually vomits two or three times directly after the onset, but after that there are often long intervals, and the vomited matter only becomes fæcal after several days. Malignant stricture is in the great majority of cases in the sigmoid flexure or the rectum, and accordingly the vomiting is late, infrequent, and only after food. Indeed, patients often say that since they took to a fluid diet they have not been sick at all. In volvulus of the sigmoid flexure, its most usual position, vomiting is of very subordinate importance, and thus contrasts with the acute character of the onset and of the other symptoms. In a case under my care the patient did not vomit at all during the whole course of his illness. When volvulus of the cæcum is present, vomiting is about as prominent as it is in intussusception.

Flatulence and rumbling of wind about the abdomen, with difficulty in the passage of flatus by the bowel, point to a partial obstruction, probably due to a stricture, a kink, or subacute strangulation beneath a band. The amount of distension and the size and situation of the distended coils of intestine will help to indicate the position of the obstruction.

When it is clear from the other signs and symptoms that a carcinoma is present in the colon, the situation of this carcinoma can be settled with a considerable degree of certainty by a careful inquiry. The patient will frequently describe how the wind invariably rumbles along a certain course in the abdomen, and is arrested at a certain point, where at the end of a wave of contraction a griping pain is felt. Sometimes he describes a mass which rises up at such times, and is dispersed by rubbing with the hands. After such a spasm he says the wind disperses upwards and is not passed. When such rumbling and pain are confined to the right loin, we may infer that the obstruction is at the hepatic flexure. When they pass up the right loin and across the upper part of the abdomen, and the pain is in the left hypochondrium, a carcinoma of the splenic flexure is usually present. When the wind rumbles down the left loin, and the pain is felt in the hypogastric or left iliac region, the obstruction is probably in the sigmoid flexure or upper part of the rectum. This latter group of symptoms is that more often encountered. *Tenesmus* indicates that the colon is implicated; it occurs in obstruction due to intussusception, volvulus, cancer of the colon, and pressure on the colon from without. The passage of *blood and slime* points to intussusception or to ulceration of the colon, which, although more often malignant in cases of intestinal obstruction, may be innocent and due to dysentery or syphilis. The two common causes of blood and slime in cases of obstruction are cancer and intussusception, which are easily distinguished by the age of the patient, and the presence of a sausage-shaped tumour in 75 per cent. of intussusceptions.

Signs—Age.—When a baby, whose anus and rectum are apparently normally formed, suffers from its birth from either a total obstruction of the bowels or a very advanced degree of constipation, it may be inferred that a congenital malformation of the intestine is present. Such malformations take the form of breaches of continuity, septa, strictures, or in the case of congenital idiopathic dilatation of the colon it is probably a diminution or absence of the nervous reflex action of defecation. It is highly probable that intestinal obstruction in a baby during the first two years of life is due to intussusception, especially if it be a male child. Meckel's diverticulum causes trouble for the most part in childhood or in early adult life. Patients with cancer of the colon are as a rule over forty, but it must always be remembered that this form of carcinoma may occur at an earlier age than any other. Fæcal accumulation is a disease of later life. The only form of acute intestinal obstruction which occurs almost invariably at an advanced age is that due to impaction of large gall-stones in the intestine. The average age of these patients, who are nearly always fat women, is about sixty years (*vide* p. 92).

The amount of *distension* is usually an indication of the position of the obstruction in the alimentary canal, and of the chronicity of the condition. Thus, the greatest degree of distension is reached in congenital constipation persisting into adult life; one such patient, who was popularly known as the "balloon man," attained to the gigantic girth of 86½ inches (Formad). Cases of acquired fæcal accumulation also show very great distension. Cancerous strictures of the rectum and sigmoid flexure frequently lead to huge distension of the abdomen. It may be stated, indeed, that great distension in intestinal obstruction always points to some cause in the colon, and that it is seldom very great unless the cause be in the lower colon and of considerable duration. A partial exception to the above statement is volvulus of the sigmoid flexure, in which the onset of distension is very acute and the abdomen becomes exceedingly tense in one or two days; but in this case the distension is not the result of slow accumulation of fæcal matter above the obstruction, but of the rapid formation of gas in the twisted intestine due to interference with its circulation. Cancer of the ileo-cæcal valve, chronic intussusceptions, simple strictures and kinks of the lower ileum, and the subacute and chronic forms of incarceration under and over a band implicating the same part of the intestine, may lead to very considerable distension of the abdomen; but in these cases the characteristic ladder arrangement of dilated small intestines is seen, although the lower coils may reach so large a diameter as to be mistaken for large intestine. Acute strangulation by bands, internal hernias, or apertures, usually occurs in the middle and upper parts of the ileum, and is seldom associated with distension until peritonitis has set in and has produced paralysis of the gut. Large gall-stones during their passage down the jejunum produce no appreciable distension. If they remain impacted in the lower ileum for several days, the abdomen may become greatly distended, especially when peritonitis has supervened.

Tumours are sometimes felt in association with intestinal obstruction; in intussusception the characteristic sausage-shaped swelling may be felt under an anæsthetic in about three-quarters of the cases, and usually in the left loin. In obstruction due to pressure upon the intestine from without, the tumour or swelling can often be felt on a careful abdominal examination, if the distension is not too great, or by the vagina or rectum when the tumour is wedged in the pelvis. Such swellings are the pregnant uterus, fibroids of the uterus, cysts and tumours of the ovary, sarcoma and cancer of the prostate, pelvic abscesses, retroperitoneal sarcoma, tumours, cysts, and abscesses of the kidney, and hydatids in various situations. Gall-stones causing intestinal obstruction have been felt in the region of the ileo-cæcal valve or in the pelvic pouch in isolated instances. Cancers of the colon can rarely be palpated on account of the greatly distended pouch above them. Volvulus and acute dilatation of the cæcum complicating a cancerous stricture of the colon can often be felt as a definite rounded swelling the size of a fœtal head, and resonant on percussion.

Contracting coils of intestine, seen through the abdominal wall and felt to harden, indicate that there is partial obstruction of at least some days' duration. Coils of normal intestine are sometimes seen slowly contracting through a greatly emaciated abdominal wall, but differ from those indicating obstruction in the absence of dilatation and of hardening as they contract.

Contracting coils of large intestine are found in the region of the abdomen appropriate to them, and they are usually over 3 inches in diameter when they harden. They are most frequently seen in the case of a carcinoma of the upper part of the rectum or the lower part of the sigmoid flexure, and are situated in that case in the left iliac fossa or the hypogastric region. It should be remembered that waves of peristalsis pass in the transverse colon from right to left. A dilated stomach frequently occupies the same region of the abdomen, but in this case the waves pass from left to right. Visible contraction of the transverse colon is, however, rare. When it does occur, it is often due to a cancerous stricture at the splenic flexure, and the waves do not then pass to the descending colon and the sigmoid flexure. The cæcum may be seen and felt dilated and contracting in some of the subacute cases of dilatation of that viscus. The malignant stricture in such cases is frequently at the hepatic or splenic flexure of the colon, although it may be in the sigmoid flexure. In most cases, however, the dilatation is so acute and the distension so great that the organ is seldom able to contract. Fæcal accumulation may give rise to a contracting colon, usually in the sigmoid flexure, which may become so greatly dilated as to occupy almost any part of the abdominal cavity, but perhaps the commonest position of the contracting coil is below and to the right of the umbilicus. The upper parts of the colon are seldom seen or felt to contract in fæcal accumulation, although they may be greatly dilated.

Contracting coils of small intestine assume the well-known ladder arrangement, and are located in the central regions of the abdomen. The largest coils, which stand out prominently at the end of a wave of peristalsis, are as a rule on the right side of the abdomen below the umbilicus. These lowest coils in chronic cases may be dilated to a diameter of 2½ to 3 inches, and it is often hard to believe that they are indeed small intestine. The cause of the obstruction is seldom lower than the ileo-cæcal valve. In other words, the small intestine is seldom seen dilated and contracting as the result of obstruction in the large intestine, although this may occur in rare instances, as shown in a case figured by Nothnagel. The great capacity of the cæcum and the tenuity of its wall appears usually to act as an efficient block between these two sections of the intestine. The causes of contracting coils of small intestine are chronic intussusception at the ileo-cæcal valve, malignant stricture of the ileo-cæcal valve and small intestine, simple strictures, adhesions, kinks, bends, and incarceration over or under a band, the latter causes being often the result of appendicitis.

A rectal examination will sometimes reveal the cause of intestinal obstruction, and should never be omitted. Cancer of the rectum may be felt or seen by means of a rectal speculum, and the sigmoidoscope reveals cancerous tumours which are far beyond the reach of the finger, but are suitable for resection. Foreign bodies, such as Murphy's button, enteroliths, fæcal masses or large gall-stones, or the lower end of an intussusception, may be felt in the rectum. Ballooning of the rectum is often associated with malignant stricture low down in the colon, in the sigmoid flexure, or upper part of the rectum. It is, however, found in peritonitis and many other conditions in which there is no true obstruction. I have seen it in acute colitis in a young man.

Enemas were formerly employed by Brinton and others to determine the position of strictures in the colon. The quantity of fluid the patient could retain was ascertained, and the position of the stricture was estimated thereby; when more than a certain amount was retained, the obstruction was supposed to be in the small intestine. Experience has shown that this method is quite unreliable. Auscultation of the colon after the administration of an enema is but little less untrustworthy.

The passage of a soft bougie is another method which has been abandoned as useless and misleading.

When the other conditions, especially peritonitis, which produce *indicanuria*, have been excluded, excess of indican in the urine on the second day of an acute obstruction shows that the small intestine is implicated, and the absence of an increase of indican after three or four days points to a cause in the colon. It is of little value as a localizing sign in chronic obstruction of the colon, for the amount is increased when the distension reaches up to the small intestine.

(iii.) **The Differential Diagnosis of Intestinal Obstruction from Similar Conditions** is sometimes a very complex and difficult matter. Intestinal obstruction is mistaken for other conditions or conversely for the following reasons: (1) All violent stimulations of the abdominal sympathetic produce the same symptoms of sickening pain, vomiting, collapse, constipation, and frequently distension. It does not matter whether that stimulation is produced by the strangulation of a coil of intestine or by a renal or biliary calculus, an acute infection of the peritoneum, or the torsion of a testis or an ovarian tumour. (2) It might be supposed that in these latter cases, as no organic obstruction exists, the bowels would remain open; but it has been abundantly demonstrated by clinical and experimental evidence that all stimulations of the abdominal sympathetic powerfully inhibit the movements of the intestine (Bayliss and Starling), and cause spasmodic closure of the ileo-cæcal valve (Elliott). Such spasmodic and paralytic forms of obstruction are sometimes most difficult to distinguish from those of organic origin. (3) Further, since the mechanism of intestinal peristalsis is most complicated and extensive, consisting of a complex neuro-muscular apparatus with an extensive vascular supply, it is not surprising that it should be liable to be thrown out of gear by the most diverse causes acting at diverse points. Thus, an embolus may block its arterial supply; thrombosis may invade its veins; some poison may act specifically upon its nerves, or these may become functionally deranged or be lacerated and stimulated by a mesenteric hæmorrhage or inflammation. In whatever way the peristaltic mechanism is inhibited, stasis follows with its symptoms of distension, vomiting, and constipation, and violent sympathetic pain and its reflex results frequently further complicate the diagnosis.

The diseases which have been mistaken for organic intestinal obstruction or conversely are innumerable, but the more important may be classified as follows:

(*a*) An organic obstruction is actually present, but for purposes of treatment it is not called "intestinal obstruction." In acute intestinal obstruction the presence of an *external hernia* must always be excluded by the routine examination of the inguinal, femoral, and umbilical rings, and by palpating the front of the abdomen to exclude a small ventral hernia passing through the linea alba, lineæ transversæ, or lineæ semilunares, or buried in a fat abdominal wall. Scarpa's triangle should be deeply palpated for an obturator, and Petit's triangle for a lumbar hernia; the gluteal region should be explored for a sciatic hernia. Strangulated hernias are almost always exceedingly tender, so that the patient indicates their position, except in the aged and in those profoundly collapsed. When the hernia is only incarcerated, however, there may be little local pain, but the severe pain from the coil of intestine may be referred to a point as high as the epigastrium in the eighth or ninth dorsal spinal areas. A patient under my care, with a small incarcerated femoral hernia concealed in a fat groin, had, in ignorance of what was the matter, actually blistered his epigastrium to obtain relief; his abdomen, which showed visible coils of intestine, was opened, and the hernia reduced. It is a serious matter to mistake *fæcal accumulation* for obstruction due to some other organic cause, since laparotomy performed for fæcal obstruction is nearly always followed by a fatal result. This mistake can nearly always be obviated by the routine practice of giving two turpentine or strong brine enemas in every case of obstruction before making a final diagnosis. The presence of compacted fæces in the rectum or putty-like tumours in the sigmoid flexure, transverse colon, or cæcum, with a history of habitual constipation and of attacks of distension and vomiting relieved by purges and enemas, should make a diagnosis of fæcal obstruction certain.

(*b*) The peritoneum has been violently stimulated by infection as in acute peritonitis due to the perforation of the appendix, stomach, or duodenum, or to tuberculous peritonitis. *Acute peritonitis* may closely resemble acute intestinal obstruction in its sudden onset with acute abdominal pain and collapse, and in severe vomiting and constipation. The vomiting, however, seldom becomes fæcal, and an enema will show that the constipation is partial. Peritonitis differs from obstruction in that the temperature is, as a rule, raised, the pulse rapid and wiry, and in the presence of the tenderness and rigidity of the abdomen. None of these signs is, however, absolute. The temperature in peritonitis may be normal or subnormal, the pulse between 80 and 90 and of good volume even in general purulent peritonitis, and even tenderness and rigidity may be absent or nearly so. As a matter of fact, however, uncomplicated cases of acute peritonitis are seldom diagnosed as intestinal obstruction, or *vice versa*. Intestinal obstruction may complicate acute peritonitis from its very onset. I have pointed out that even in mild attacks of appendicitis the last coils of the ileum may be so inflamed, kinked, or twisted and fixed, as to produce complete intestinal obstruction from the commencement of the attack. On the other hand, many cases of intestinal obstruction are complicated by peritonitis. In strangulation of intestine from whatever cause, peritonitis is usually present after the third or fourth day. Cancerous stricture of the colon frequently terminates acutely by perforation either just above the stricture or in the cæcum. Such mixed cases of intestinal obstruction and peritonitis are exceedingly difficult to diagnose correctly.

Tuberculous peritonitis in its ordinary forms is not difficult to distinguish from obstruction, but in children there may be acute and transient forms which are usually mistaken for intussusception; possibly they are due to the rupture of a caseous tuberculous gland in the mesentery. The patients are delicate children from three to ten years of age, in whom, after a period of ill-health lasting about a week, there is a sudden onset of severe abdominal pain, constant vomiting, and constipation often resistant to purges and enemas. The tongue is moist and creamy, the temperature about 100° F., and the pulse good. The abdomen is tumid and slightly distended, not tender, and with the stethoscope the intestine can be heard to contract. Free fluid can often be detected in the peritoneal cavity, and there may be palpable glands in the abdomen. A turpentine enema is usually effective, and the little patients rapidly convalesce. Tuberculous peritonitis may produce acute obstruction by the formation of peritoneal or omental bands, or by general matting and adhesions.

(*c*) The abdominal sympathetic system has received a violent sensory stimulus, as in *torsion of the testicle, ovary, or omentum, or in the passage of a biliary or renal calculus.* It has been pointed out that the symptoms of all violent stimulations of the abdominal sympathetic are, during the first few hours, almost identical whatever the cause may be. The history will, however, often assist in a diagnosis, and as the case progresses differential signs appear. In torsion of an undescended testis the organ is missing from the scrotum, and may be found at the internal ring or in the inguinal canal. In a twisted ovarian tumour there is often a history of a previous swelling in the abdomen, and the engorged tumour is nearly always palpable. Moreover, the symptoms of internal hæmorrhage, such as blanching, restlessness, and a wiry pulse, are often present when the tumour is large or has ruptured. In the case of a twisted ovary, testis, or omentum, a turpentine enema will be followed by the passage of some flatus and fæces. In biliary colic there is often a history of previous attacks, and the distribution of the pain is more or less characteristic, and the pain may be followed by jaundice. The same remarks apply to renal colic, in which alterations in the urine, especially hæmaturia and frequency, are seldom absent. This class of case does not often cause any difficulty in diagnosis after the first twelve or twenty-four hours.

(*d*) The sympathetic nerves in the mesenteries have been violently stimulated, as in *acute pancreatitis and hæmorrhage into the mesentery*. The pancreas lies at the root of the mesentery and transverse mesocolon, and is in intimate relation with the cœliac plexus and semilunar ganglia. In many cases the destructive necrosis and hæmorrhage spread between the layers of the mesenteries rather than to the peritoneal cavity. Many attacks of acute hæmorrhagic pancreatitis are apyrexial. The irritation of the sympathetic ganglia and plexuses produces severe pain, collapse, vomiting, and a small and feeble pulse. The invasion of the roots of the mesenteries irritates the branches of the splanchnic nerves to the small intestine and colon, and these become distended and their movements inhibited, so that almost total constipation results. In acute pancreatitis, however, the pulse is extraordinarily weak compared with the other symptoms; the temperature is often raised; a tender swelling may be felt deep in the epigastrium, and an enema usually produces a result. It may be said that when the peritoneum is not much invaded in acute pancreatitis it is more often than not mistaken for acute obstruction. Glands or hydatids in the mesentery may become acutely inflamed, or hæmorrhage may occur into that structure under exceptional conditions. My colleague, Mr. Roxburgh, performed laparotomy on a child with the symptoms of intestinal obstruction with melæna, and found hæmorrhage into the mesentery. Next day the child developed a hæmorrhagic smallpox rash.

(*e*) Some irritation of the gastro-intestinal mucous membrane is present, as in arsenical and other forms of *poisoning*, and in some epidemics of *cholera*, *enteric fever*, and *colitis*. Acute strangulation has often been mistaken for poisoning, especially by arsenic, when the attendant circumstances were suggestive; and in some instances, as in three cases quoted by Leichtenstern, the true state of affairs has only been discovered at the autopsy. During epidemics many cases of very acute obstruction have been diagnosed as cholera, especially when characterized by anuria and death early in the disease. The mistake is still more easy when diarrhœa is present with obstruction, as is very rarely the case. A recent typhoid epidemic in the East End of London was characterized by an acute onset with profuse vomiting, severe colic, and constipation. Many cases were apyrexial, or nearly so at first. Several cases were diagnosed as acute appendicitis, and one at least was explored as possibly a case of intestinal obstruction. Typhoid spots appeared later in this case, and a positive agglutination reaction was obtained.

Simple colic due to the ingestion of indigestible articles of diet in adults or in children has often been mistaken for acute internal strangulation. The vomit, however, is never fæcal, nor the constipation absolute, though enemas may be retained from the shortage of fluids in the body. Acute colitis in children may readily be mistaken for intussusception from the association of vomiting with collapse and the passage of blood and mucus, especially when a tumour be felt in the abdomen. In one case of this kind, in which I opened the abdomen, the tumour turned out to be the right kidney covered by peritoneum on three sides. In other cases the contracted sigmoid flexure may be palpable, but its diameter is less than that of an intussusception. In colitis the temperature is almost constantly raised from the start, the onset is not so sudden, and bile is found upon the napkin with the blood and slime. In acute intussusception, after the first motion or two, pure blood and mucus are passed, and the temperature is, as a rule, not raised until the second or third day. Cases of chronic intussusception are almost always diagnosed as chronic colitis, at least for a time; fæcal matter is mixed with mucus and blood in both cases. The presence of a sausage-shaped tumour in intussusception is the diagnostic sign, but I have known the decision delayed until the ileo-cæcal valve presented at the anus.

(*f*) Some toxic cause has acted on the neuro-muscular mechanism of the intestine, as in lead-poisoning and uræmia. To avoid mistaking *lead colic* for acute intestinal obstruction, the gums should be examined for a blue line, and inquiry made as to the trade of the patients. The subjects of lead colic are usually painters, white-lead workers, or have habitually handled old corroded lead, and many give a history of previous attacks in themselves or their fellow-workmen cured by castor-oil and opium. An enema is usually successful. It must be remembered, however, that lead-workers may suffer from intestinal obstruction, appendicitis, and peritonitis, and that their previous history is then very misleading.

Uræmia may closely simulate either acute or chronic obstruction. The points of similarity are profuse and urgent vomiting, well-marked constipation, pain in the abdomen and back, collapse, and not uncommonly, in the chronic form of uræmia, distension. Careful examination of the patient and of the urine should lead to the recognition of uræmia. The mental condition is, moreover, usually more obscured in uræmia, and suggests a toxic condition. If coma be present a mistake can hardly occur. I have seen acute dilatation of the upper colonic segment, with profuse vomiting and total constipation, in chronic uræmia. In another case a pregnant woman with acute nephritis and anuria was operated on for profuse vomiting, complete constipation, and severe abdominal pain. The gynæcologist and surgeon had overlooked the anuria.

(*g*) The cause of the derangement of the neuro-muscular apparatus of the intestine is organic or functional nervous disease, as in *tabetic crises, hysteria, and other obscure nervous diseases.* The alimentary canal is divided into segments by sphincters: (1) The pyloric, (2) the ileo-colic, (3) a sphincter probably in the region of the splenic flexure, and (4) the sphincter ani. The innervation and functions of these segments have been so far differentiated that it is not surprising that their functional derangement should be more or less distinguishable. The toxic conditions which simulate organic obstruction probably act through these neuro-muscular mechanisms. Acute uræmia acts chiefly on the gastric segment, lead colic mainly on the enteric segment, whilst acute dilatation of the upper or lower colonic segments is seen in chronic uræmia. The purely nervous conditions which simulate organic obstruction show indications of a similar distribution. Tabes in gastric crises attacks the gastric segment, and also produces severe constipation, often associated with loss of tone in the sphincter ani, which is probably a derangement of the lower colonic segment. I performed laparotomy for profuse vomiting, complete constipation, and severe attacks of abdominal pain, in a syphilitic and alcoholic man who suffered from lightning pains in the legs, and was more or less insane. The following parts of the alimentary canal were found distended: the stomach, lower half of the ileum, and the colon, as far as the splenic flexure. The rest of the intestine was contracted, the transition from the dilated to the collapsed intestine being abrupt. No organic cause of obstruction was found, and purgatives and enemas were subsequently effective. Acute dilatation of the upper colonic segment in otherwise healthy people has been described, associated with vomiting and complete constipation, and has led to colotomy in the transverse colon. The nervous derangements of the intestine here described are closely allied to acute dilatation of the stomach.

(*h*) The intestine is extensively paralyzed by *embolism or thrombosis of the mesenteric vessels.* The abdominal signs and symptoms of embolism of the superior mesenteric artery and acute thrombosis of the portal vein are very similar. In thrombosis, however, a considerable quantity of blood-stained fluid is found in the peritoneum, and the spleen is frequently enlarged. The history and associated signs of the cause must mainly be relied on to distinguish them. In embolism mitral stenosis or infective endocarditis is probably present, but the abdomen is normal until the absolutely sudden onset of the attack. In thrombosis there are signs of some cause, such as cirrhosis of the liver, cholelithiasis, or thrombosis of other veins, and the acute attack is preceded by obscure abdominal pains and transient attacks of diarrhœa and vomiting. In their final and acute attack both conditions resemble intestinal obstruction, in their sudden onset with severe pain followed by vomiting, which is violent, and soon becomes brown in colour. Distension is present, and the bowels are often completely constipated for the last day or two. On the other hand, the attack may commence with diarrhœa and often with melæna. Hæmatemesis is common, and there is usually a considerable quantity of free fluid in the abdomen. In the three cases of thrombosis and one of embolism that I have seen, organic intestinal obstruction was suggested by the symptoms, and in one it was actually diagnosed. Laparotomy was performed in two cases, and all the patients died.

(*i*) The intestine is not obstructed, but vomiting and constipation are present, and a contracting viscus or a tumour is found in the abdomen, as in pyloric obstruction, cancer of the greater curvature of the stomach, or the contracted form of carcinoma of the stomach. These three forms of carcinoma of the stomach may simulate chronic obstruction of the transverse colon. Pyloric obstruction resembles chronic intestinal obstruction in the presence of obstinate constipation. The bowels are often closed for seven days at a time, and then small hard scybala are passed. Purgatives fail to act, for they are retained in the stomach and provoke further vomiting. Vomiting is present at irregular intervals, and frequently occurs after food, but its great quantity and fermenting character should direct attention to the dilated stomach. In pyloric obstruction an elongated organ in active peristalsis may be found at almost any level in the abdomen, and may be mistaken for the transverse colon, but in gastric peristalsis the waves pass from left to right. By giving a teaspoonful of sodium bicarbonate, followed by the same quantity of tartaric acid, the outline of the stomach can be made visible.

Cancer of the greater curvature of the stomach, especially in a case of gastroptosis, may be mistaken for cancer of the colon. If the growth does not interfere with the orifices or movements of the stomach, gastric signs and symptoms may be almost absent. Moreover, carcinoma in this situation tends to spread along the great omentum to the transverse colon, and may irritate or actually obstruct that organ. In a case under my care, the passage of mucus, blood, and scybala, associated with a mobile tumour in the left iliac fossa and absence of vomiting, led to a diagnosis of carcinoma of the sigmoid flexure. A laparotomy showed that the tumour was carcinoma of the greater curvature of a greatly displaced stomach, and that the transverse colon was surrounded by the growth. The form of carcinoma known as "india-rubber bottle stomach" may irritate or obstruct the colon in a similar way, but the contracted stomach is concealed beneath the liver and ribs, and cannot be examined either by palpation or percussion after distension with gas. Moreover, the gastric condition produces persistent vomiting of small quantities. Such cases are seldom diagnosed correctly.

5. TERMINATIONS, DURATION, AND PROGNOSIS.

Terminations.—Death is certain to follow in practically every case of organic intestinal obstruction which is not submitted to operation, with the exception of those due to fæcal accumulation. It may occur early in the attack, and is then due to collapse. In acute strangulation it is most often due to peritonitis, the result of a crude perforation or of the passage of micro-organisms through the intestinal walls. Many patients succumb to toxæmia or to septicæmia, and, where fæcal vomiting has occurred, not a few die of inhalation broncho-pneumonia. Very rarely death has been brought about by phlegmonous processes and suppuration in the liver and lungs.

Duration.—A case of acute intestinal obstruction may terminate within a few hours, or last as long as twenty-one days. The average duration of 350 acute cases collected by Leichtenstern was six days. Chronic obstruction may drag on for months, or even years, and in fæcal accumulation absolute constipation has persisted for as long a period as eight or nine months (Strong).

Prognosis.—*Spontaneous cure* may occur in any form of intestinal obstruction, but this is so exceedingly rare that it cannot be anticipated. It is more often seen in fæcal impaction than in other forms of obstruction, and it certainly occurs in cases of compression of the intestine by tumours from without. Large gall-stones and foreign bodies are sometimes passed even after days of obstruction; occasionally in invagination the intussusceptum will slough away safely; and in both volvulus and internal hernia spontaneous recoveries have taken place.

Recurrence is a very unusual occurrence. It is more common in cases of volvulus and intussusception. The opinion that intussusceptions frequently recur is derived from the time when this condition was treated by forcible inflation. The intussusception was then only partially reduced. In recent years the majority of cases have been treated by laparotomy, and direct reduction and recurrence is almost unknown; thus, one case only has recurred at the London Hospital in recent times. The recurrence of symptoms may be due to an entirely new cause of obstruction, as in a case in which I performed colotomy for carcinoma of the colon, and some days later acute obstruction supervened, due to strangulation of a coil of ileum by an omental band.

Mortality of Intestinal Obstruction after Operation.—More than half the cases of intestinal obstruction submitted to operation die. Of 669 cases of all forms of intestinal obstruction treated in the surgical wards of the London Hospital from 1893 to 1905, 366 died and 303 recovered, a mortality of 54·7 per cent. I am indebted to Dr. Holzmann for the following table showing the mortality of a series of 100 consecutive cases of acute intestinal obstruction treated from 1900 to 1902 inclusive at the London Hospital:

	Cases.	Died.	Recovered.	Mortality.
				Per Cent.
Strangulation by bands or through orifices	34	16	18	47·0
Intussusception	52	28	24	53·8
Volvulus	8	7	1	87·5
Impacted gall-stones	6	4	2	66·6
	100	55	45	55

These figures are derived from hospital records, and therefore are nearer the truth than statistics of published cases, which are as a rule not consecutive. It should, however, be emphasized that, of the 55 fatal cases in the above series, no fewer than 29 were admitted to the hospital collapsed after several days' obstruction; 26 of these died of shock soon after the operation, and 3 were not operated upon, as they were moribund. If it were fair to assume that all these cases would have recovered if operated on earlier, the mortality of this series would fall to a little over 25 per cent. It may be stated, therefore, that when recognized early, and operated on within two or three days of the onset, the mortality of acute intestinal obstruction is about 30 per cent., or 1 case in 3, and that the mortality of chronic intestinal obstruction in similar circumstances is not higher than 15 per cent.

The following points bear on the prognosis of intestinal obstruction: (1) The age of the patient and his general vitality; (2) the duration of the illness; (3) the presence of collapse, toxæmia, or septicæmia; (4) the presence of peritonitis; (5) the presence of distension; (6) the cause of the obstruction, if this can be ascertained.

Age.—In babies and the aged the prognosis is bad. This is well shown by the following series of 100 acute cases analyzed by Dr. Holzmann:

Age.	Cases.	Recovery.	Death.	Mortality.
				Per Cent.
Under 1 year old	38	15	23	60·5
Over 50 years old	14	5	9	64·3
Over 1 year and under 50 years ...	48	25	23	47·9
	100	45	55	55

Age, no doubt, accounts for the high mortality in intussusception (53·8 per cent.), and in gall-stone intestinal obstruction (66·6 per cent.).

Distension in acute intestinal obstruction of the small intestine is a most serious sign. It indicates not only that the operation will be difficult, but the probable existence of peritonitis, and the presence of a large quantity of toxic matter which must be evacuated before the patient is free from the danger of toxæmia. I have never seen a case of intussusception with distension in a baby recover. In chronic obstruction distension is not of such serious import.

The cause of the obstruction has an important bearing on the prognosis. The prognosis is probably worst in volvulus, while impacted gall-stones and intussusception are very serious forms of intestinal obstruction. Strangulation by Meckel's diverticulum is also very fatal, for gangrene of the intestine and peritonitis appear early in such cases.

In short, if the patient be a young adult with healthy viscera, and has not been ill more than two or three days, the prognosis after operation is good. On the other hand, if the patient be old and feeble, with a fatty heart, granular kidneys, emphysema, and the illness has lasted several days, the prospect is wellnigh hopeless.

6. The General Treatment of Intestinal Obstruction.

Excluding fæcal impaction, the treatment of organic intestinal obstruction is purely surgical, and will therefore be referred to here in the briefest manner only. The objects the surgeon has in view are to open the abdomen, determine the cause of the obstruction, and relieve it with the least possible loss of time. The median *incision* below the umbilicus is usually chosen, because through it practically all parts of the abdomen can be reached. To locate the cause of the obstruction, the following method is usually adopted: The cæcum is first examined; its distension indicates that the cause is in the colon. The sigmoid flexure is next drawn out; distension shows that the obstruction is in the upper part of the rectum or lower part of the sigmoid flexure. Should the sigmoid flexure be collapsed, the transverse colon is drawn into the upper angle of the wound; if this be distended, the obstruction will be found at the splenic flexure or in the descending colon. If the transverse colon be empty whilst the cæcum is distended, the cause of obstruction will probably be found at the hepatic flexure. When the cæcum is found to be collapsed, the obstruction is in the small intestine. The surgeon then as a rule draws out the collapsed coil of ileum entering the cæcum, and traces it backward to the obstruction. On the other hand, he may prefer to draw out a distended coil of small intestine, and, having determined which is its lower end, follow it down to the cause of the obstruction.

The special treatment adapted to each cause of obstruction cannot be discussed here. The general principle which guides the surgeon when he has located and identified the cause is to relieve the obstruction at once, and to remove the cause, if this can be done in a few minutes. Thus, a band may be divided, a gall-stone evacuated, or an intussusception or volvulus unravelled. If, however, the removal of the cause would take some considerable time, as in the case of a carcinoma of the colon, the point of obstruction must be short-circuited by lateral anastomosis, or the gut opened and drained above it. The cause may then be dealt with at a second operation, should the patient recover from his intestinal obstruction. It would be wrong to leave the subject of the treatment of intestinal obstruction without insisting that opium should not be given to an acute abdominal case until a definite diagnosis has been made or the patient has finally refused operation.

PART II.—SPECIAL FORMS OF INTESTINAL OBSTRUCTION

SYNOPSIS.—1. Causes within the lumen of the intestine—internal obturation: (i.) True foreign bodies, (ii.) enteroliths, (iii.) fæcal masses, (iv.) gall-stones. 2. Causes in the wall of the gut—internal stricture: (i.) Carcinomatous stricture, (ii.) cicatricial stricture, (iii.) congenital stricture; congenital idiopathic dilatation of colon. 3. Causes outside the intestine: (i.) Anomalous forms of adhesions, (ii.) compression, (iii.) herniaform intestinal obstructions due to (*a*) bands, (*b*) apertures, (*c*) internal hernias. 4. Intussusception. 5. Volvulus of the intestine; volvulus of the omentum.

1. INTERNAL OBTURATION OF THE INTESTINE.

CASES of this class of intestinal obstruction may be subdivided as follows: Those due to (i.) true foreign bodies, (ii.) enteroliths, (iii.) fæcal masses, (iv.) gall-stones.

(i.) **Intestinal Obstruction due to True Foreign Bodies**—*Frequency.*—Foreign bodies are an extremely rare cause of intestinal obstruction; only one case occurred in the 669 consecutive cases of obstruction at the London Hospital during the years 1893-1905.

If we include foreign bodies arrested above strictures and bands, four further cases are recorded, although no doubt, if all the post-mortem records of cancers of the colon and rectum were reviewed, a number more might be added to this class. In the great majority of these, however, these is no proof that the foreign bodies—fruit-stones for the most part—were impacted in the stricture.

Etiology.—The circumstances under which foreign bodies are swallowed are very various, interesting, and bizarre.

Children in their years of total inexperience show an instinctive tendency to convey to their mouths and swallow every suitable object they encounter in their crawling expeditions. Coins, pins (simple and safety), brooches, pieces of coal, "comforters," corks, beads, and many other objects, are thus devoured. G. G. Roy[1] tells how a child swallowed a mouse, and suffered from severe gastro-intestinal symptoms in consequence.

When years of discretion are attained, the ordinary human being only swallows foreign bodies by accident, as when a tooth-plate is ingested during sleep or under anæsthesia, or a false eye is swallowed in a glass of water. The ordinary careless individual, however, swallows with his food fish, fowl, rabbit, and other small bones, and the stones of such fruits as plums, cherries, prunes, dates, and apricots. Perhaps these latter should not be classed as accidents, for Nature provided the luscious epicarp in order to induce some animal to swallow the stone and widely distribute the seed suitably manured. Accordingly we find the stone is shaped to pass safely along the alimentary canal, and it is only when the fruit is eaten in vast quantities, or the stones are arrested by a stricture or some other abnormality of the alimentary canal, that any trouble is caused by them.

Burglars and thieves, in their endeavour to conceal the products of their nefarious deeds, have swallowed coins, watches, rings, jewels, and other objects.

Jugglers form a special class, for the foreign bodies ingested by them are very large or very numerous, or of a strange and unsuitable character, in order to excite the wonder, admiration, and generosity, of their audience. Jugglers may be divided into two classes: Those who pass down their œsophagus large objects, such as dinner-knives, swords, etc., which are only accidentally allowed to slip beyond reach; and those who will really swallow pence, pipes, watch-chains, studs, pocket-knives, corks, string, straps, and any other small strange object, for a few pence, for an audience in some low public-house or as a result of a wager.

As an example of the first of these varieties of juggler may be mentioned the corroded remnants of a large table-knife which form Specimen 2377[2] in the Royal College of Surgeons Museum. Two months before his death one William Dempster swallowed at Carlisle a table-knife 9 inches long. He had passed it into his œsophagus, and was holding the tip of the blade between his teeth, awaiting a suitable opportunity to remove it, when some practical joker slapped him on the back and the knife slipped. He wandered the country seeking for relief, and died vomiting blood-stained fluid continuously. The remains of the knife were in the stomach, which was not perforated. The horn handle had been digested, and a large part of the steel blade and haft. It should be noted, however, that he died apparently of gastritis, and not of obstruction.

The second class of juggler provides us with many more examples. Everard Home[3] had under his care at St. George's Hospital a sailor who for a bet swallowed three clasp-knives. He safely passed the blades, springs, and rivets, having digested the horn parts of the handles, and recovered. The Human Ostrich[4] was another remarkable example. He was a man aged forty-two, who was accustomed to go from one public-house to another in the East End of London and amuse the people by swallowing coins, etc., and eating bottles, plates, and pipes, for money. He was admitted to the London Hospital in 1894, under the care of Mr. F. S. Eve, suffering from acute peritonitis. Two perforations were found in the lower part of his ileum, 3 inches apart. Through the one projected a piece of tinfoil, and through the other a hook attached to a piece of leather. Eighteen inches of bowel was blocked with foreign bodies. The bowel was stitched to the abdominal wound.

At the post-mortem the following objects were found in the stomach and intestines: Twenty-five pieces of cork, twenty pieces of tinfoil, a leaden bullet, a piece of leather 9 inches long with a hook at either end, several pieces of pipe-stem and portions of newspapers, an iron ring, a rubber ring, a piece of string 1 foot long with pieces of tinfoil and cork tied at intervals upon it.

W. S. Halsted[5] has related an even more remarkable instance of a collection of articles swallowed by a man. He combined the trade of carpentry with juggling, and was twenty-one years old. Four days before admission to the Johns Hopkins Hospital he had been challenged in a public-house, by a crowd of American medical students, to swallow, whilst stripped to the skin, articles provided by them. This he did for seven hours, from 7 p.m. on Saturday evening until 2 a.m. on Sunday morning. He was seized soon after with vomiting and pain. Professor Halsted opened his abdomen, and as the mass of metal prevented the stomach from being drawn out of the wound, he made a small opening in it, and sewed around the edge of this a funnel-shaped piece of linen, walling off the general peritoneum with gauze.

Two and a half hours were expended in extracting the following 208 articles:

20 pieces of small dog-chain (460 cm.).
1 piece of large dog-chain (29 cm.).
4 watch-chains (31 cm.).
1 brass chain (59 cm.).
2 pieces of chain (15 cm.).
10 horseshoe nails.
54 wire nails (16 of these 7½ cm. long).
35 ordinary nails (8 of these 6 cm. long).
8 screws (2½ to 3 cm.).
2 screw-eyes.
7 knife-blades.
1 knife-handle.
50 tacks.
12 pins.
1 piece of tin.

The wound broke down and gut protruded, but the man ultimately made a good recovery.

Hysterical girls provide a large number of extraordinary cases of foreign bodies ingested. The articles devoured include keys, safety-pins, ordinary pins, nails, needles, hair, finger-nails, skeins of wool and cotton, and many other objects.

A girl attended the out-patients of the writer, stating that she had been startled whilst holding an open safety-pin in her mouth, and had swallowed it. Her abdomen was skiagraphed, and was found to contain the open safety-pin, another closed, a door-key, some coins, and other objects. She was directed to eat porridge and avoid purgatives, and to return to the hospital if she experienced any inconvenience, or when she had determined to discontinue her bad habit.

Mr. Mayo Robson[6] has recorded a case of an intelligent girl, aged ten, who suffered from gastric symptoms, the cause of which was revealed when she vomited a garden nail.

He removed successfully from the stomach 42 cast-iron garden-nails 1⅝ inches long; 93 brass and tin tacks from ½ to 1 inch long; 12 large nails, some brass-headed; 3 collar-studs; 1 safety-pin; 1 sewing-needle. During twenty-two days following the operation the patient passed *per anum* and vomited 30 garden nails, a piece of a needle, 1 stud, 8 tacks, and a pen. Even after her recovery this patient could not abstain from satisfying her morbid appetite with indigestible articles.

A desire to eat their own hair seems also to characterize some hysterical women. Some pull them out, others cut the ends off. The Royal College of Surgeons Museum contains two such specimens.

A girl[7] aged twenty complained of a large upper abdominal swelling. On three occasions, five years previously, she had inexplicably become bald. She could eat an ordinary meat diet, and was not seriously ill.

A roll of human hair 12 inches long, and weighing 5 pounds 3 ounces, was extracted from the stomach. She recovered.

Another specimen[8] consists of three human hair-balls, one of which was passed *per anum* by a woman aged twenty-two. She had suffered from vomiting, constipation, and abdominal pains, and her death was apparently due to peritonitis. At the autopsy the two other hair-balls were found in her stomach. One of these weighed 10½ ounces. The hair was of the same shade as her own, which had been noticed to grow short on several occasions.

Hair is quite indigestible, and has a tendency to mat into masses, as is shown in the well-known bezoars of animals.

When the patient has possessed in addition the habit of biting the nails, these have been found added to the mass of hair.

The most remarkable case of pin-swallowing is, perhaps, that reported by John Marshall.[9] The patient was a woman with a keen appetite, who proceeded from starch and slate pencils in her youth to bent pins in her middle life. She had hæmatemesis on two occasions, and when thirty-six years old vomited without ceasing for days, but slowly recovered. When forty-two, after three weeks of incessant vomiting, she died. In the pyloric portion of the stomach 9 ounces of bent pins were found, and in the duodenum nearly a pound of the same. The intestine below the mass was contracted, so that apparently the gut was obstructed. Several pins ready bent to be swallowed were found in and around her death-bed.

Lunatics are especially prone to devour the most unsuitable of objects, partly from a desire to commit suicide, and partly to satisfy their morbid appetites. Many such cases have been recorded, and include such things as spoons, forks, egg-cups, wood, clothing, cocoanut matting, leather, and hair.

Thus a woman aged fifty years,[10] who had for ten years resided in a lunatic asylum, possessed an inveterate propensity to lick and gnaw wood. She had recovered on one occasion from an attack of œsophagitis due to the impaction of a splinter of wood in the gullet. She died of chronic gastritis, and at her autopsy a ball of woody fibres and splinters, 9 inches long and 2½ inches wide, was found moulded in the pyloric portion of her stomach, which was enormously dilated so as to be ½ inch thick, chiefly by increase of the circular fibres. The ball was of a dark colour, and was the shape of the stomach. The stomach was covered with chronic inflammatory papillomata.

Certain trades appear to lend themselves to the swallowing of foreign bodies, which are either of moderate size in themselves, or which, by accumulation and matting, produce balls or masses in the stomach or intestines. Thus dressmakers are prone to swallow pins or needles held in their mouths, or small portions of thread they have bitten off. Carpenters frequently carry nails in their mouths and inadvertently swallow them, and in some trades, where the fingers are frequently wetted in the mouth, small objects are constantly swallowed and may collect. The following is a very remarkable case of this sort: A boy was employed in plucking chickens, and constantly wetted his fingers in his mouth. He thus ingested enough down to form around a fæcal centre a calculus the size of a large hen's egg. He suffered from melæna and repeated attacks of colic for nine months. A tumour was then located in his right hypochondrium, and Dr. Alexander[11] removed the above mass by enterotomy in 1882. The patient recovered.

Since the introduction of Murphy's button for intestinal anastomoses, several cases have been recorded in which these buttons became impacted.

The following is a good example of this surgical accident:

Isabella O., a woman aged forty-five, was suffering from carcinoma of the stomach with hour-glass contraction. On July 3, 1903, Mr. F. S. Eve performed gastro-enterostomy with a Murphy's button at the London Hospital. She did well for a fortnight, and then developed signs of partial obstruction of the small intestine, colicky pain, vomiting, and partial constipation. These signs having persisted for six days, and a radiography having shown the button to be situated in the middle of the pelvis, Mr. F. S. Eve opened the abdomen and removed the button from the lower ileum. The patient did well.

Pathology and Symptoms of Intestinal Obstruction due to True Foreign Bodies—The Cause of the Extreme Rarity of Obstruction as a Result of Foreign Bodies.

The narrow places in the alimentary canal are—

(i.) The œsophagus: (1) At the level of the cricoid; (2) where the left bronchus crosses it; (3) where it pierces the diaphragm; (4) at the cardiac sphincter.
(ii.) The pyloric sphincter.
(iii.) The termination of the ileum and the ileo-colic sphincter.
(iv.) The anus.

The œsophagus at the level of the cricoid is the smallest of these. It therefore comes about that, if a body passes the œsophagus and pylorus, it will almost certainly negotiate the ileo-colic orifice without much difficulty; and once in the colon, nearly all are discharged without trouble or delay. In other words, the œsophagus and stomach act as a filter, arresting all those foreign bodies which might otherwise have caused intestinal obstruction.

Even when this is appreciated, there is still room for wonder at some of the objects which pass safely through the small intestine, until we study closely the beautiful muscular mechanism for passing bodies along the alimentary canal.

Peristalsis is usually regarded as a ring contraction, which by its progress down the gut pushes the contents in front of it; and if this were all, it would indeed be difficult to understand how an open safety-pin or a plate of teeth could be pushed along the twenty feet or so of small intestine without arrest or perforation.

The longitudinal fibres of the intestine, however, play a simultaneous and equally important part. When the circular fibres have contracted behind the object so that it cannot pass backwards, the longitudinal fibres draw the coil of gut below over the body as a glove is drawn on to a hand or a stocking on to a foot. The progress of contents is therefore rather the sleeving of the tube over the contents than the propulsion of the contents along the tube.

Concerning the Foreign Bodies which cause Obstruction.—The great *variety* of these has been indicated sufficiently already.

Size: The larger the body, the more likely is it to obstruct; thus knives, forks, and egg-cups, are nearly certain to do so.

Number: Some foreign bodies—such as fruit-stones, water-melon seeds—appear to cause trouble only when present in great numbers. Apparently the intestine is not able to propel more than a certain mass of weight, and when this is exceeded, not only does the mass remain in some dependent loop, but further additions are made to it.

Thus Dor[12] narrates a case where a mobile tumour in the right iliac region was associated with constipation, vomiting, and finally peritonitis. The patient died, and the ileum just below the ileo-cæcal valve was found dilated into a huge sac which contained a conglomerate mass of 120 prune-stones and 92 leaden bullets. The latter had been swallowed, apparently, to remove the obstruction, but they had precipitated the fatal ending.

Some quite minute bodies—such as vegetable hairs, animal hairs, down of feathers, threads of cotton and wool, and bent pins—produce large masses by their capacity for matting together. Such cases will be more fully treated under Concretions, but an instance of a mass of bent pins has already been quoted, and another case in which the obstruction was due to matted down from fowls' feathers.

The *shape* of the foreign body also has an important bearing. When rounded and smooth, large objects may safely traverse the small intestine. As an example of this, we may instance the vast majority of Murphy's buttons employed in gastro-enterostomy. Even Murphy's buttons, however, are not always innocuous, as has already been narrated.

Fish and other bones, needles, pins (especially open safety-pins), and plates of teeth with projecting wires, are bodies obviously adapted to impaction, and it is only remarkable how often they traverse the whole length of the alimentary canal in safety.

Fruit-stones are adapted by their ovoid shape to pass without hindrance along the intestine.

The changes which foreign bodies undergo in their passage form a most interesting subject.

(1) *Incrustation.*—Organic bodies tend to become incrusted with fæcal matter and phosphates, and may thus grow to be considerable concretions. On the other hand, inorganic bodies have little tendency to incrustation.

Mr. Childrens' case[13] illustrates this difference. A lad aged nineteen swallowed some plum-stones whilst eating oatmeal daily and at every meal. For three months he suffered from colic, vomiting, and the constant passage of watery stools containing blood. He wasted rapidly and died. Three concretions the size of tangerine oranges were then found in the arch of the colon, in a pouch. One which was bisected contained a plum-stone in its centre. The rest of the mass was formed of the setæ from the oat-seeds matted around this stone.

Sir Frederick Treves[14] has also described an enterolith he removed from a patient who had persistently taken magnesia, which substance formed the mass of the concretion, but the nucleus was formed by a gall-stone.

(2) *Solution* is of great importance, for some foreign bodies tend to dissolve, become blunted, or break up into smaller parts, which may be safely evacuated, whilst others are completely resistant to the alimentary juices, and though small in size may accumulate in masses which are practically indestructible. Epidermal structures are almost completely indigestible, and to this fact we owe the formation of hair-balls, or bezoars, and those which in addition contain portions of finger-nails and wool.

Bone is only very slowly digested, and solution exerts practically no influence on large masses of bone. Of vegetable substances, cellulose and xylen—the material of which wood and nutshells are formed—are scarcely acted on at all in the alimentary canal. Indeed, Nature has covered many seeds in a xylen sheath in order that they may pass through the intestines of animals without being digested, and starch granules are often enclosed in a cellulose cover in a similar way. Hence the importance of boiling starchy food, to burst this capsule, before it is taken as a food.

Of metals, iron is acted on most readily, copper to a much less degree, lead scarcely at all, and gold is quite unaffected.

The sharp points of needles and the edges of knives are soon blunted and rounded.

The motions often become black from the iron salts they contain, and this may indicate the cause of otherwise misleading symptoms.

Bryant[15] has narrated of a woman of forty-three that she swallowed thirteen iron screws or bolts, each 2½ inches long, with a raised collar ¾ inch in diameter and a square head. When swallowed they weighed 24 ounces of iron. The first was passed in forty-one days, and the last in six months. She then recovered. The screws lost 4¾ ounces in weight in transit.

Velpeau[16] has recorded the following case: A man swallowed a bone, and afterwards an iron fork. The latter remained in his stomach fifteen days, the prongs pointing to the left. It then passed the pylorus, and occupied six weeks in traversing the small intestine. For five months it lodged in the right flank, and during this time he suffered from colic and black stools, alternating with constipation. The fork then broke in half, the prong half of it was passed *per anum*, and he recovered.

(3) *Germination.*—A curious fact of not much practical importance is that seeds may germinate in the alimentary canal.

Thus, Landais[17] tells of a man who passed in his stools 400 cherry-stones, several of which had sprouted.

H. H. Rodebaugh[18] has recorded the same phenomenon in the case of beans.

The Rate of Progress of Foreign Bodies along the Alimentary Canal.—In this respect they may be divided into three classes:

(i.) Those that pass with the fæcal stream without interruption.
(ii.) Those that are arrested for longer or shorter periods, but ultimately are evacuated.
(iii.) Those that become permanently arrested.

(i.) *Those that pass with the Fæcal Stream without Arrest.*—It is fortunate that these form the vast majority of all bodies that are ingested. Only a minority cause any trouble at all, and then it is in the œsophagus and stomach. Of the very few that cause trouble in the intestines, the greater number produce perforation and peritonitis, or abscess formation, and even among these true obstructive symptoms are very rare. It is therefore not surprising that only one case of pure obstruction due to a foreign body has occurred at the London Hospital in thirteen years, out of a total of 669 cases of obstruction due to all causes; and that case, which has already been recorded, was due to a Murphy's button, employed in a gastro-enterostomy, which became impacted in the lower ileum. Foreign bodies take from two to thirty days to pass through the alimentary canal even when they are not actually arrested or impacted.

Symptoms are usually absent altogether, but there may be transient attacks of colic, which when severe may lead to mild vomiting in susceptible patients.

Innumerable cases have been recorded belonging to this class, but the following illustrate the variety sufficiently:

A baby ten weeks old was admitted to my wards at the London Hospital with an open safety-pin impacted at the level of its cricoid. In the efforts made to close and withdraw the pin, it was pushed into the stomach. Gastrotomy was contemplated, but the child passed the pin, still open, in forty-eight hours. It was then found that the point of the pin had been providentially turned over inwards before the pin was swallowed. A hernia curiously complicated a similar case reported by Boardman.[19] A ruptured child swallowed a nail $2\frac{1}{2}$ inches long. In a few days the nail was felt in the rupture, and in due course was passed at the anus.

In another instance an infant ten months old swallowed half a brass toy locket with sharp edges, measuring $\frac{7}{8}$ inch and $\frac{3}{4}$ inch. It impacted in the pharynx, and was pushed down by the father to prevent suffocation. This object was passed in thirty-six days little altered and without notable symptoms (F. W. E. Kinnier[20]).

Marcé[21] tells of a lunatic woman who in order to commit suicide swallowed a doubled-up silver pencil-case 8 centimetres long. She expelled it in eighty-six hours without it having produced any obvious effect.

Finally, a girl fell whilst being pursued by her lover, and broke a knife she was carrying in her mouth, and swallowed the blade, 7 centimetres long. She bled profusely from the pharynx, but the blade, black and corroded, was passed in four days without other ill effect (Schmidt[22]).

(ii.) *Those Foreign Bodies that are arrested for Longer or Shorter Periods, but ultimately are evacuated.*—These bodies are as a rule of an irregular shape, and in their passage become disposed crosswise in the intestine. The common site of impaction is in the termination of the ileum, or actually at the ileo-cæcal valve or in the cæcum, so that the signs and symptoms are as a rule situated in the right iliac fossa.

Varying degrees of inflammation are produced in the intestine during the arrest of the body. Acute enteritis frequently results, with pyrexia, colicky pain and tenderness, tympanites, and constipation, which gives place after some days to serous fœtid stools. When ulcerative enteritis supervenes, the symptoms become worse, constipation and diarrhœa alternate, and pus and blood may be found in the stools. Finally, the inflammation may reach the peritoneum and produce subacute localized peritonitis with adhesions. In such cases there will be well-marked rigidity and resistance felt in the abdomen at the site of the arrested body.

The period occupied by this class of foreign bodies in traversing the alimentary canal is usually a matter of months, but it has extended to as long a period as ten years.

Thus Planque[23] reports the case of a Spanish officer who swallowed a fork whilst scratching the back of his tongue with it. He passed it 454 days later without any symptom of note.

Gosselin's[24] patient was not so fortunate. He was a juggler aged thirty years, who swallowed a pipe 10 centimetres long, which contained no tobacco, but had been smoked on four occasions. He suffered from severe colic, vomiting, and diarrhœa, and there was dulness and resistance in the right iliac fossa. He passed the pipe intact two months after he had swallowed it, but the symptoms continued, and he died five days later. His colon was found to be extensively ulcerated.

In yet another instance a female melancholic lunatic swallowed three teaspoons. Her symptoms were pyrexia, bilious vomiting, and constipation, which was total during three days. Her abdomen was distended and painful, especially in the right iliac fossa. After three days she suffered from profuse diarrhœa, and her symptoms abated. She had four such attacks of obstruction, and then she passed the three spoons matted into one mass by fæces and covered with mucus. She recovered.[25]

Velpeau's case has already been alluded to, where a fork remained fifteen days in a man's stomach, five weeks in his intestine, and lodged for five months in the right flank. It then broke in half, and he passed the prongs and recovered.

(iii.) *Those Foreign Bodies which become permanently arrested.*—The causes of permanent arrest of foreign bodies may be classified as follows:

(1) The ileo-colic sphincter and termination of the ileum.
(2) Strictures of the intestine, simple or malignant.
(3) Herniæ.
(4) Diverticula and pouches.
(5) Bands and adhesions.
(6) Loops.
(7) Tumours pressing on the gut from without.

(1) *The Ileo-Colic Sphincter and Termination of the Ileum* are in many respects similar to the anus and rectum. In both there are a sphincter and detrusor singularly liable to spasm (and this is, moreover, the only narrow isthmus in the intestines). For these reasons foreign bodies are arrested here.

(2) *Strictures and Foreign Bodies.*—The strictures of the intestine which lead to the arrest of foreign bodies are either innocent or malignant. They are situated either in the small intestine or in the colon. The vast majority of recorded cases have been malignant strictures of the lower colon.

The bodies arrested have been most various, and include fruit-stones of all kinds, tea and cabbage leaves, currants, raw potatoes, worms, and many other objects.

The foreign body often occludes the narrow opening of the stricture, and leads to the onset of acute obstruction. In the case of the small intestine, owing to the fluid nature of its contents, there may have been no previous symptom of any sort.

The following very remarkable case illustrates this well, and was under the care of my colleague, Mr. James Sherren:[26]

Simon S. was a Polish Jew aged thirty-six. He ate fifteen raw potatoes for a bet, and was soon after seized with vomiting and constipation, and later with abdominal distension and dulness in the left flank. Mr. J. Sherren opened his abdomen, and found the raw potatoes impacted above a stricture in the upper ileum. He performed enterotomy with a Paul's tube. The patient was of a very savage and wild disposition; he cursed his nurses, tore off his dressings, and on three several occasions small gut prolapsed through his wound. Nevertheless he made a good recovery after the fistula and the stricture had been excised and the ends of the intestine united.

I have seen the most acute intestinal obstruction produced by the impaction of a mass of vegetable leaves in a malignant ring stricture of the colon. This case is reported under the heading of Cancer of the Colon.

In the case of cancerous strictures of the colon, however, there have usually been premonitory symptoms of partial obstruction. These suddenly become acute after the ingestion of the foreign bodies.

Thus my colleague, Mr. T. H. Openshaw, had under his care a woman aged thirty-three, who had suffered from chronic obstruction, with the passage of blood and slime, for a year or more. After eating freely of currants she was suddenly seized with total obstruction, distension, and later with vomiting. Left inguinal colotomy was performed, and a large quantity of currant seeds and skins were irrigated from above the stricture. She did well.

On the other hand, bodies may collect above a stricture without seriously affecting the fæcal stream.

Such a case has been put on record by Clement of Shrewsbury.[27] A colotomy of the ascending colon was performed for a narrow stricture of the transverse colon, which was apparently malignant. The woman did well for a time; then 116 plum-stones and two bones were evacuated from above the stricture.

Post-mortem records abound with such cases. In some instances the foreign body appears to have assisted in perforation of the intestinal wall above the stricture.

(3) *Herniæ and Foreign Bodies.*—A foreign body is very liable to be arrested in a hernia and lead to incarceration, strangulation, gangrene, or perforation. In many instances the body passes safely through, as in the case of the ruptured infant who swallowed a nail, already related. The following illustrates strangulation due to a foreign body:

A long-standing hernia became acutely strangulated in a cook. He refused operation until *in extremis*. His sac was then laid open, and the foot of a lark was found fixed in the strangulated coil (J. L. Petit[28]).

In a similar case recorded by Farcy of Fleche,[29] a porter had suffered from a reducible hernia for eight years. This became suddenly strangulated, and was so hard that Farcy dare not reduce it. He bled the man for several days. The hernia then suppurated, and was incised. Sixteen mutton bones were removed from the gangrenous loop, and the fæcal fistula closed in thirty-three days.

(4) *Diverticula, Pouches, and Foreign Bodies.*—Foreign bodies are often permanently arrested in diverticula. Such diverticula are the cæcum and its appendix, and pouches formed specially for the lodgment of the body. The latter pouches are either symmetrical dilatations of the lumen of the gut or they are lateral bulgings.

When a foreign body has entered a pouch or diverticulum, it shows little tendency to cause obstruction unless it is subsequently displaced after it has grown in size from fæcal and phosphatic concretions.

Such a foreign body is, however, exceedingly prone to produce ulceration, gangrene, and perforation, but this does not concern us here.

(5) *Bands and Foreign Bodies.*—In just such a way as a stricture will arrest a foreign body passing down the intestine, so will a band compressing the gut from without.

An instance of a gall-stone so arrested is given under the heading of obstruction by these bodies.

In the following very remarkable case an intestinal worm (*Ascaris lumbricoides*) was so arrested:

A girl aged twenty-two was admitted to the London Hospital, under Mr. E. Hurry Fenwick, with signs of acute peritonitis. On opening the abdomen, an intestinal worm was found loose among the intestines. A search was made for the point of perforation, and it was found immediately above the spot at which a band constricted the gut. The band was divided, the perforation closed, and the peritoneum washed out. She recovered.

The Results of Permanent Arrest of a Foreign Body in the Intestine.—These results are—(1) Obstruction; (2) enteritis and ulceration; (3) perforation.

(1) *Obstruction.*—Pure obstruction of the normal intestine is, apparently, the rarest of all results produced by true foreign bodies, if we exclude all concretions and calculi, which grow continuously larger until they do produce obstruction. Enteritis, ulceration, and perforation, are much more common terminations of such cases, although to these is added some partial obstruction. However, some large smooth bodies are capable of causing true obstruction, as in Mr. Denby's case.[30] A man aged sixty years had had a reducible hernia for twenty-five years. Three months before his death he suffered from diarrhœa. Three weeks before his death he had severe abdominal pain and vomiting. A week later the symptoms returned, and he had in addition fæcal vomiting, hiccough, and distension, but, curiously enough, his bowels were frequently moved by small fluid motions. The hernia was reduced without much difficulty, but his symptoms did not improve.

At the autopsy a large "willow pattern" egg-cup was found impacted in and perforating the lower ileum a short distance above the hernia. The gut above was greatly distended.

(2) *Enteritis and Ulceration.*—The intestine above and around an impacted foreign body is often profusely ulcerated. Indeed, it is not unusual for the body to lie in one huge ulcer.

It is the presence of these ulcers which obscures the diagnosis of obstruction, for their discharge leads to frequent fluid stools, which, however, contain pus if carefully examined. Pyrexia and colic may be attributed directly to the ulcer, and also the local tenderness over the impacted mass.

Wasting and marasmus are a consequence partly of the obstruction and partly of the profuse discharge.

(3) *Perforation.*—This matter does not really concern us, but it is added for the sake of completeness.

In some cases the bodies perforate without symptoms of peritonitis, and wander to distant parts of the body. This is especially so in the case of needles, and many marvellous cases have been recorded by Planque, Birth, Silvy, Villars, and Otto.[31] The last will suffice as an example:

A woman, Rachel Hertz, displayed the most complex hysterical signs, including mania, convulsions, coma, paralysis, incontinence, retention, and aphasia. She also discharged needles from all parts of her body up to the number of 395.

There is, however, no evidence that she swallowed them. This, indeed, is the weak point in all these cases of needles, etc., removed from distant parts of the body, and supposed to have been swallowed. Some years ago I was asked to scrape out a chronic patch of supposed lupus on the dorsum of the foot of a boy. I discovered a small aperture in the deep fascia, and in exploring this found a stiff pig's bristle rather more than an inch long beneath the fascia. The chronic inflammation then subsided. There is, however, no evidence that the boy had swallowed it.

In other instances, however, the proof is clearer. A woman died after having been ill for twenty-three days with hectic fever and a double phlegmasia alba dolens. At the autopsy a needle was found projecting into her vena cava, around which a thrombus had formed (H. Thompson[32]).

In another instance (Velpeau[33]) a pin was found projecting from the ascending colon to the right ureter, so that its head was in the one and its point in the other. An abscess had formed around it. A case has been recorded where an ear of grass was swallowed by a child, and discharged on its back. As a rule localized peritonitis and adhesions are established before the perforation, and an abscess forms into which the body projects. Such abscesses are, of course, familiar objects in connection with appendix concretions, but foreign bodies may be safely extruded from any part of the alimentary canal in a similar way.

The following are examples of the safe passage of such foreign bodies through the abdominal wall by adhesion and suppuration.

Dr. Lettsom[34] tells of a person who wished to provoke vomiting, and for that purpose swallowed a cobbler's knife. It subsequently issued from below his ribs.

A little girl aged five had a pain and swelling below and to the right of her umbilicus. An abscess burst in sixteen days, which discharged pus for eight months. Then fæcal matter appeared in the discharge, and two months later the point of a hair-pin, which was manipulated out. She had no recollection of swallowing it (S. Jones[35]). This last case is even more surprising. A lunatic woman having swallowed the plug of her bath-tub, and passed it with impunity, proceeded to ingest a pair of scissors 5 inches long. An abscess formed to the right of her umbilicus, and when this burst the point of the scissors protruded. The aperture was dilated, and afterwards the rivet was removed and the blades extracted separately. The fistula closed in two days (H. B. Sands[36]).

In other cases the foreign body is not extruded through the skin, but passes into some other organ.

Thus a piece of rabbit bone has been known to pass from the intestine into the bladder, and a pin has perforated the ureter from the colon. Other foreign bodies have perforated into the vagina, vena cava, and liver.

Adhesions have not always formed when perforation occurs, and acute perforative peritonitis then supervenes, as in the case of the noted Ostrich Man already referred to. In yet other and rarer instances the foreign body slowly leaves the intestine, and becomes buried in dense inflammatory tissues, in the centre of which a small collection of pus is often found surrounding the foreign body. Such masses are often mistaken for sarcomata or other neoplasms.

My colleague, Mr. T. H. Openshaw, encountered such a case. A large dense mass had slowly appeared in the right flank, and was suspected to be a sarcoma, but on incision a steel fork was found to occupy an abscess cavity in the centre of it. The wound was drained and the patient recovered.

Prognosis in Swallowed Foreign Bodies which have reached the Intestine.—It must be reiterated that the risk is slight, and that the vast majority of such bodies which reach the intestines are safely evacuated.

Large and sharp bodies, such as knives, forks, spoons, and scissors, may reach the intestine, however, and the risk in such cases is very great. Even small bodies, such as pins, grapestones, and shot, are dangerous to life when they lodge in the appendix.

Perforation with general or local peritonitis is a most serious complication.

Death occurs in the fatal cases of foreign bodies in the intestines from these causes and in this order:

1. Marasmus and wasting from ulceration and enteritis, leading to diarrhœa, vomiting, and septic poisoning. This condition may continue even when the foreign body has been expelled, as has been sufficiently illustrated.
2. Peritonitis and abscess formation.
3. Least often is it due to uncomplicated intestinal obstruction.

Treatment of Swallowed Foreign Bodies.—It cannot be too often repeated that nearly all foreign bodies which have passed the pylorus are naturally evacuated sooner or later. Their safe passage down the alimentary canal is said to be facilitated by coarse food, such as porridge and vegetables, and an avoidance of fluids and purgatives; but there is no doubt that even on this regimen the contents of the small gut would still be fluid.

Even when the foreign body has become impacted, probably in the region of the ileo-colic sphincter, it is still better to wait a day or two. A careful watch should, however, be kept on the temperature, pulse, and tumour, so that interference may be undertaken before ulceration or perforation has been produced.

There is no doubt that the tendency of modern surgery is to needlessly interfere with foreign bodies which have been swallowed, without waiting to see if they will cause trouble. A skiagram is taken, and dramatically demonstrates some foreign body in the stomach. The surgeon cuts down on it next day, and finds that the foreign body has already left the stomach. It is probably in the rectum. A long and useless search follows, greatly to the detriment of the patient. Skiagrams for such purposes should be taken as short a time as possible before operation. Even then it is most difficult to be certain in which portion of the intestines the body is placed.

If the intestine is not adherent, an incision may be made on the foreign body, and this may be removed and the aperture closed. Where there are numerous bodies, or they are of such an irregular shape that immediate extraction does not seem possible, the intestine may be fixed in the abdominal wound and opened. The gut may be resected and sutured when the bodies have been safely evacuated.

Should an abscess exist, this should be opened and drained like an appendix abscess. Should an irregular-shaped foreign body be found in its centre, this should be removed with the greatest possible caution and circumspection. The exit of a foreign body should never be hurried.

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(ii.) **Intestinal Concretions, Calculi, Enteroliths, etc.**—*Frequency.*—This condition is exceedingly rare: one case has occurred in 669 cases of intestinal obstruction at the London Hospital in thirteen years.

Etiology and Pathology.—The essential conditions which govern the formation of intestinal concretions are as follows:

1. *A nucleus*, which is nearly always of an organic nature; such

nuclei are fruit-stones, masses of vegetable or animal hairs, or gall-stones.

2. *A cause of arrest* is a necessary condition. The vermiform appendix frequently arrests such a nucleus, or a saccule in the colon or cæcum. The lower end of the ileum is another favourite site for arrest, probably in consequence of its dependent position in the pelvis or as a result of adhesions from appendicitis or salpingitis.

3. *Adhesive particles* must be supplied continuously in the food. These particles are sometimes thread-like structures (such as hairs and wool) and vegetable fibres (such as cotton, hemp, the setæ of the oak-seed). Nearly all these threads have a spiral structure, which enables them to interlock.

Other particles adhere by a process of crystallization. Such are magnesium and iron salts, salol and other drugs.

4. *Chronic enteritis* must be started and maintained around the growing concretion. The chronic irritation provides one organic element and a colloid menstruum in which crystallization readily takes place. The decomposition of proteids by bacterial action produces ammonia phosphates and carbonates; and if calcium or magnesium salts are present, triple phosphates are readily produced.

Intestinal concretions, calculi, or enteroliths, may be classified as follows:

1. Those concretions which are formed from some habitual element of the food which progressively accumulates. Oat, fat, starch, and meat-balls, are examples.

2. Concretions formed from amorphous fæcal matter.

3. Those composed of minute foreign bodies frequently swallowed, such as thread, string, hairs, and wool.

4. Certain drugs persistently taken may produce concretions, such as magnesia and iron salts, gum benzoin, liquorice root, salol, and amalgams administered for worms.

5. Calculi, which are hard like stones, and are composed of concentric layers of phosphates and carbonates of calcium, magnesium, and ammonium, with an organic base. These are almost entirely the result of the chronic inflammatory processes set up by the lodgment of a nucleus in an intestinal diverticulum or pouch.

6. Mucous balls have been described as a cause of intestinal obstruction. These were probably cases of membranous colitis.

7. Balls composed of the *Ascaris lumbricoides* have also been

described, but it is very doubtful if they ever occlude the intestine.

Symptoms.—The symptoms of a progressively enlarging concretion differ somewhat according to its location in the ileum or the colon.

(*a*) *The Concretion is in the Ileum.* — The history is one of successive and progressively severer abdominal attacks. In the earlier attacks the symptoms are rather those of enteritis, colic, pyrexia, diarrhœa, and a variable degree of vomiting. As the condition progresses, obstructive symptoms, such as distension, visible and contracting coils of gut, severe vomiting and constipation during the attack, begin to be more prominent. A tender swelling may also be found in the right iliac region, or on a rectal or vaginal examination. As the case goes on, and actual ulcers are formed around the stone, pus and blood appear in the stools. The final attack of complete obstruction is probably due to a displacement of the concretion from a pouch into the lumen of the gut, or to spasm of the intestine due to an acute attack of enteritis.

(*b*) *The Concretion is in the Colon.*—The symptoms are those of attacks of ulcerative colitis, severe colic, tenesmus, and the frequent passage of watery stools containing pus and blood. The patient wastes progressively. A tender swelling is more often palpated than when the concretion is in the small intestine. Obstructive symptoms are rare, and only occur very late in the disease.

Treatment. — This condition is seldom diagnosed before an operation or an autopsy. Should the concretions occupy the rectal pouch, they may be broken up and evacuated. The sphincter ani may require to be forcibly dilated for this purpose. In higher situations the treatment is purely surgical. The concretion is displaced upward from the region of ulceration, if this is possible. The intestine is then incised and the mass removed.

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(iii.) **Intestinal Obstruction due to Fæcal Masses** (**Fæcal Accumulation or Coprostasis**).—The final stage of habitual constipation is the formation of fæcal tumours in the colon, which block its lumen and produce attacks of intestinal obstruction.

Incidence.—Of 669 consecutive cases of intestinal obstruction which were admitted to the London Hospital in thirteen years, 69 were due to fæcal impaction.

Etiology.—Fæcal accumulation is a disease of the colon. It cannot be reiterated too often that pathology is only disordered physiology, and that the only sure foundation of knowledge in any disease is a thorough acquaintance with the anatomy of the organ involved, and of its normal functions. The functions of the great gut are, unfortunately, still shrouded in much mystery.

The Physiology and Anatomy of the Colon.—Everard Home[1] was one of the earliest workers at this subject. In his lectures on comparative anatomy delivered in 1814, he recorded some experiments with extract of rhubarb. After he had swallowed a large dose of this drug, it appeared in his urine in an hour. It then disappeared for two or three hours, to reappear at the end of that period. The conclusion he drew from this observation was that absorption was rapid in the upper and lower parts of the alimentary canal, and he surmised that the points at which this absorption took place were the stomach and the cæcum. He also was the first to point out the striking correlation between the stomach and the cæcum, and he inferred that the latter possessed important digestive functions, which were developed inversely as those of the stomach. In ruminants, in whom the stomach is large, the cæcum is small; whereas in such other herbivora as the horse and rabbit, in whom the stomach is small, the cæcum is greatly developed. Everard Home also examined the contents of the colon in ducks. He was able to extract a considerable quantity of fat from the contents of the cæcum, but from the fæcal matter of the lower colon he could obtain none.

For many years after Everard Home nothing further was added to our knowledge of colonic functions. Comparative anatomists taught, from a study of the cæcum in herbivora, that cellulose was digested there, and it followed of necessity that the products of this digestion were absorbed in the colon below.

When the great work of Pasteur revealed the enormous part played in Nature by bacteria, and workers came to investigate the micro-organisms of the alimentary canal, it was discovered that these performed an essential part in the digestion of cellulose, which substance is not acted on by any of the secretions of the digestive glands. Schottelius[2] reared chickens in a sterile cage upon sterilized food, and found that they wasted away and starved in a few weeks; but when supplied with food containing normal micro-organisms they rallied, and were restored to health. With the exception, however, of its cellulose digesting function, which was thought to be unimportant in man, the colon was regarded merely as a receptacle for the refuse products of the small intestine, where fluids were extracted, and where the débris was compacted into fæces, and could wait a convenient time to be expelled from the body.

The doctrine that the colon in man was no better than a cesspool, and was only tolerated in the animal kingdom at all in creatures who had to fly from swift foes, and could not stop to evacuate their excreta, culminated in the teachings of Metchnikoff.[3] He claimed that old age was a disease due to the chronic action of toxins on the specialized cells of the body, more especially the cerebral, renal, hepatic, and the yellow elastic tissue which gives elasticity to arteries. When these cells were sufficiently damaged, they were devoured by phagocytes and replaced by connective tissue. Metchnikoff taught that these toxins were derived from three chief sources: from syphilis, from alcohol—which, after all, is only the toxin of the yeast micro-organism—and, since people grew old and died who had not had syphilis nor imbibed alcohol regularly, Metchnikoff attributed these cases of degeneration to the absorption of toxins produced by the profuse "bacterial flora" which flourishes in the great gut. He urged that in all men products of bacterial action, such as phenol, indol, and skatol, were normally present in the urine; that they increased greatly in constipation, and especially in intestinal obstruction, but disappeared in cases in which a fistulous opening had been made in the ileum, to return when that opening was closed.

These considerations led Metchnikoff to foretell the time when man would have his colon excised in order to prolong his life to 140-180 years, which he regarded as the allotted time of man, founding this opinion largely on the age of the patriarchs as recorded in Genesis. Metchnikoff's aversion to organs which he regarded as vestigial led him further to attack the stomach, which he thought was very much better deleted from the alimentary canal as well. He regarded the small intestine as sufficient by itself.

During the last few years the activity of surgery in connection with the appendix has caused a great revival of interest in the functions of the colon.

Dr. W. B. Cannon[4] introduced bismuth into the intestines of a cat, and was then able to observe the movements of the colon through the intact abdominal wall by means of X rays. He thus demonstrated that the ileum discharged its contents into the colon, which by retroperistaltic waves starting in the transverse colon threw them back into the cæcum, where constant churning movements indicated active digestive processes, until finally a forward wave of peristalsis emptied the contents into the lower colon.

Dr. Barclay Smith[5] confirmed these observations, and found that when the roots of the sacral nerves were stimulated in the cat and dog the whole colon was constricted, but that in the rat, which is omnivorous, only the part of the colon below the left kidney was thrown into spasm. From this observation it would appear that in the rat, and probably in man, who is also omnivorous, the cæcum and the ascending and transverse colons are engaged in active digestive work, but the descending colon, sigmoid flexure, and rectum, are mainly containing and expulsive in function.

Professor Arthur Keith,[6] approaching the subject from the anatomical side, has shown that the ileo-cæcal valve is a sphincter which regulates the entrance of intestinal contents into the colon, and that there is another sphincter in the ascending colon which retains them there whilst they are submitted to processes of digestion. These sphincters he has demonstrated in man.

Mr. T. R. Elliott[7] has worked out the innervation of the ileo-colic sphincter, and demonstrated that it is as definite in function as the sphincter ani.

Sir William Macewen,[8] in his Huxley address, 1904, reviewed the literature of this subject, and recorded many interesting observations of his own. A patient of his had had a part of his abdominal wall blown away by an explosion, and through the hiatus the mucous membrane of the cæcum, the ileo-cæcal valve, and the orifice of the appendix, could be clearly seen. Just before the ileo-cæcal valve opened to discharge its acid contents into the cæcum, a viscid alkaline fluid could be seen pouring from the orifice of the appendix and from the cæcal glands. Pawlow has demonstrated an acid reflex in the duodenum which controls the pyloric sphincter. A similar acid reflex probably exists in the cæcum and controls the ileo-colic sphincter, so that the contents of the cæcum are never normally more than neutral. In Sir William Macewen's case this function appeared to become disordered, and the cæcum was now and then flooded by corrosive acid chyme which digested the skin around the fistula. From this observation he inferred that a normal function of the cæcum was to neutralize the acidity of the chyme and to destroy the ferments contained in it. I may add that a comparison of cæcotomy openings with other colotomy fistulæ confirms this view, for corrosion and digestion of the skin is only seen in the former, and is absent even in openings into the transverse colon. I would also suggest that some forms of chronic ulcerative colitis may be due to loss of this power of the cæcum to neutralize the chyme and destroy its ferments.

Sir William Macewen thought it probable that one of the functions connected with the lymphoid structure of the appendix was to maintain an attenuated culture of bacillus coli, and to inoculate each charge of chyme as it entered the cæcum.

Etiology of Habitual Constipation.—From the above review of what is known of the functions of the colon, it will be seen that these functions are most important, but that there is much left to be elucidated, nor can we clearly indicate the disorder of function which leads to habitual constipation and atony of the colon. We may, however, reasonably infer that diet, the manner of feeding, and exercise, are at fault.

The colon was evolved in primitive man to digest coarse food, consisting mainly of raw fruit and vegetables. In such food the starch granules are often enclosed in cellulose coverings, and these were removed in the cæcum. We may further surmise that primitive man was forced to active exercise in hunting for his food, and, when he was successful, ate it with relish and with hunger.

The modern town-dweller is provided with a highly nitrogenous diet, from which raw vegetables are almost entirely excluded. His food is ground fine and cooked, so that the cellulose casings of the starch granules are burst by inbibition. He swallows his food in haste, and often without appetite or attention. He is no longer compelled to regular exercise in the daily search for food, so that his lower colon is not stimulated by regular and vigorous contractions of the abdominal wall to expel its contents.

It is really no matter for surprise that under such a change of circumstances the colon should become inert, relaxed, and distended with the decomposing refuse of food.

It would be out of place to enter fully into the etiology of habitual constipation, for which books devoted to medicine must be consulted, but the following classification of the main causes may not be out of place :

1. *Alimentary Constipation* includes those cases in which meat forms the major part of the diet, and vegetables, especially raw fruits, are almost entirely excluded.

The digestion of vegetables leads to the formation of gas, and their fermentation to the production of acids, and both of these stimulate intestinal movements.

Thus Rubner[9] found that carrots passed along the alimentary canal in 4 hours, brown bread in 14 hours, white bread in 17 hours, and animal food in 3 to 4 days.

2. *Lack of Exercise* and a sedentary life.

3. *Mental Work.*

4. *Irregularity* in emptying the rectum. The influence of habit is extremely well marked in obtaining a regular evacuation of the bowel. Constipation is particularly prone to occur among people of irregular habits, and has often dated from a break in regularity, such as may arise during long journeys or prolonged illnesses.

5. *Muscular Atrophy of the Colonic Wall.*—Nothnagel[10] has described cases in which the wall of the colon was only 0·12 millimetre to 0·25 millimetre thick, as against the average of 0·5 millimetre to 1·0 millimetre. This condition may be of congenital origin, or acquired as the result of long-continued colitis or overdistension. In rare cases the constipation of cases of chronic tuberculosis and alcoholism may be attributed to fatty degeneration and atrophy of the muscle of the intestinal wall.

6. *Nervous Causes of Constipation.*—Under this heading must be included such conditions as insanity, hypochondriasis, neurasthenia, hysteria, and organic lesions of the brain and spinal cord. In recurrent melancholia constipation frequently comes on and passes off with each attack. Some writers, by a confusion of cause with effect, have described all these conditions, with the exception of organic lesions, as symptoms and consequences of constipation. Constipation can only produce such results when the patient is already strongly predisposed to nervous disease.

7. *Mechanical Causes.*—Virchow has always been quoted as the authority for the mechanical theory of habitual constipation. It is true that, at autopsies on some very advanced cases of fæcal accumulation, adhesions, kinking of the flexures, anchoring of the gut, partial volvulus, shortening or lengthening of the mesentery, and abnormal positions of the cæcum, transverse colon, and sigmoid flexure, have been found, which indubitably interfered with the evacuation of the colon.

Even Virchow, however, recognized that these were probably the result of previous stages of constipation, and therefore could not be regarded as primary causes of constipation.

Moreover, as Nothnagel says,[11] " In reality, anchoring of the intestines by peritoneal adhesions is relatively rare, as compared with the extraordinary frequency of habitual constipation."

8. *Weakness of Striated Skeletal Muscles.*—Constipation is very common in the emaciated and in those conditions in which the abdominal wall is at a disadvantage, such as in pregnant women and multiparæ. Diastasis of the recti muscles and large herniæ act in a similar way.

9. *Circumscribed Atony or Spasm* has been regarded as the determining course in some cases of constipation. The latter are, apparently, those cases in which opium acts as a laxative, as it does in lead colic with constipation. Belladonna has a similar action in these cases.

10. *Pain* may lead to constipation, not only by the very natural disinclination to defecation, but also by producing spasm of the sphincter ani and associated inhibition of movements in the colon. Such pain is produced by piles, fissure and ulcers of the anus, inflamed ovaries and Fallopian tubes, and a chronically inflamed appendix. From the above list such organic causes as strictures, pressure of tumours, foreign bodies, and malformations, have been omitted, for they are treated of elsewhere.

. *The Morbid Anatomy and Pathology of Coprostasis.*—We are not concerned with constipation before the stage at which more or less permanent fæcal masses occupy the colon.

1. *The Fæcal Tumours.*—Fæcal masses may be single or multiple, and in the latter case there may be two or three large masses or a chain of small ones in the sigmoid flexure, producing a condition resembling a rosary.

Fæcal masses are most often formed in the sigmoid flexure, and next in order of frequency in the rectum. The cæcum is a common site for them, and then the flexures of the colon or its transverse loop. In this connection it should be remembered that in the normal colon solid fæces are only found in the sigmoid flexure and the rectum. In size these masses may vary from that of an egg to a huge cast of the whole dilated colon.

The latter form of fæcal mass is formed by a progressive accumulation from above upon a primary fæcal tumour in the rectum or the sigmoid. The shape of fæcal tumours may be rounded and nodular, beaded or elongated. They may be angular in outline or shaped like bricks.

Their consistency varies from that of putty, impressionable by the examining finger, to a stony hardness.

The hardness of a fæcal mass is proportional to its age, for all plastic masses of matter tend to become compacted as time goes on, under the continuous action of gravitation between their component parts. In this way fluid is expressed from them, and in time they become dry and very hard.

2. *Lengthening of Mesenteries and Changes in Position of the Colon.*—One effect of the continuous residence of masses of fæces at various points along the colon is a tendency to drag that part down into the pelvis, and in this way to lengthen its mesentery. The meso-sigmoid may become 6 or 8 inches long, and its points of origin may be so approximated as to readily admit of volvulus. In other cases the loaded sigmoid, by falling into the pelvis, produces a kink at its junction with the rectum.[12] I have seen a cæcum with a meso-cæcum 10 inches long, which allowed it to pass beneath the mesentery and to reach the region of the spleen, where it had undergone axial rotation. In a similar way the loop of the transverse colon may be dragged down into the pelvis. This displacement is liable to produce kinking at the hepatic and splenic flexures. In other cases the kink is at the centre of the loop. The colon in such cases has been compared to the letters **M**, **U**, or **V**. It is evident that these kinkings of the colon will assist in the production of obstruction. When large fæcal masses, in dependent portions of the colon, occupy the pelvis, they press upon the rectum, especially during efforts at defecation, and thus increase the difficulty of evacuating the bowel.

3. *Shortening of Mesenteries and Anchoring of the Gut.*—It is a common experience, when performing inguinal colotomy, to find the meso-sigmoid so short that the gut cannot be drawn out of the wound. This condition has received several explanations. When any abdominal organ becomes distended, it expands between the layers of its mesentery, and thereby shortens it. The inflamed and septic mucous membrane of the coil involved is supposed by others to set up a chronic sclerosing process around the lymphatics of the mesentery, and this, as time goes on, contracts. Others have attributed the anchoring of the sigmoid flexure and other parts of the colon to a progressive formation of adhesions from the base of the mesentery to its attachment to the gut. Such adhesions are not commonly seen in autopsies on cases of chronic constipation.

Fixation and anchoring of the colon is supposed to add further difficulty to the progress of fæces along the gut, but this is probably not an important factor. In many forms of adhesive and sclerosing peritonitis the gut is matted into an inextricable mass, yet there is not usually any interference with the progress of its contents.

4. *Stercoral Ulceration of the Colon.*—When fæcal masses have remained in the colon for a long time, they produce around and above them ulcers in the mucous membrane.

The position of these ulcers depends upon that of the fæcal masses. Thus, they are common in the rectum and sigmoid, but the cæcum appears to exhibit an unusual predisposition to them. In size they vary from that of a shilling or half-crown to a huge ulcerating surface entirely surrounding the fæcal tumour. Their extent and severity bear no relation to the size of the accumulation. Some enormous masses are associated with only small and scattered ulcers, whilst a relatively small fæcal tumour causing no distension or obstruction may rest in a huge foul ulcer.

In some cases they are mere superficial excoriations; in others they are deep sloughing ulcers involving the muscular or even the peritoneal coats, and leading in rare instances to gangrene of the gut. The presence or absence of stercoral ulcers, and their degree, are the main factors in determining whether constipation seriously affects the health of the patient or not.

The effects of stercoral ulcers are of three kinds: (*a*) Discharge; (*b*) absorption; (*c*) peritonitis and perforation.

(*a*) *Discharge.*—This may be serous, purulent, or sanious. It is largely responsible for the attacks of diarrhœa which punctuate the progress of cases of habitual constipation, and sometimes lead to continuous incontinence.

(*b*) *Absorption* occurs from the raw surfaces of the ulcers. The result of this absorption will depend on the virulence of the micro-organisms and toxins which are absorbed. In the most severe cases the patient may suddenly die of intestinal septicæmia, or slowly succumb to intestinal toxæmia. In slight cases there is little more than malaise, a loss of energy, a slight degree of anæmia, and some loss of flesh. In others the main effect of absorption is attacks of "fæcal fever," and this is particularly prone to occur in children.

When the nervous system of the patient is of an unstable or neurasthenic type, the absorption of these toxins may produce a sense of pressure and heaviness in the head, vertigo, somnolence, stupor, hypochondriasis, or even attacks of mania and insanity.

(*c*) *Peritonitis and Perforation.*—As the ulcers extend in depth, localized peritonitis is set up around the colon, and adhesions are produced which bind it down and anchor it. When the ulcer is infected with a virulent micro-organism, sloughing and perforation occur. Should adhesions have formed previously, a localized abscess results, which may be either intraperitoneal or placed in the retroperitoneal cellular tissue.

On the other hand, when perforation occurs into the open peritoneal cavity general peritonitis is produced.

Mr. James Berry has recorded such a case.[13] A man aged seventy-three years was admitted to St. Bartholomew's Hospital, collapsed, and died soon after. He had been constipated for years. The present attack of obstruction had lasted nine days. The sigmoid flexure was found enormously enlarged, and adherent to the liver and spleen by old adhesions. Several ulcers were present, and these had ruptured, producing general peritonitis. The rest of the colon was normal, and no stricture was present.

When the infection of the ulcer is exceedingly virulent, or when that part of the colon becomes greatly distended, large areas of the intestinal wall may become gangrenous around each ulcer. This is particularly prone to occur in the cæcum.

Mr. Southam[14] has reported the case of a man aged sixty-seven who had suffered from chronic intestinal obstruction which terminated with general peritonitis. The cæcum and ascending colon were found packed with hardened fæces. A stercoral ulcer had perforated the cæcum.

5. *Distension and Pressure Effects.*— In advanced cases the abdomen may become hugely distended by accumulated fæces and flatus. The intestines, liver, and stomach, are pressed upon and their functions interfered with. When the accumulation is mainly situated in the rectum and sigmoid flexure, the liver and stomach may be entirely forced over into the upper and right parts of the abdominal cavity. After long-continued distension, and especially in cases of congenital constipation, the lower ribs become bent out and the subcostal angle is rendered more obtuse. The thoracic organs are displaced upwards. The heart has been found, at an autopsy on such a case, situated behind the manubrium sterni, and its apex-beat has been felt in the third intercostal space. The lungs are compressed and forced upwards. The dome of the diaphragm may reach as high as the second rib on the left side and the fourth rib on the right side. The symptoms of such compression of the thoracic organs are palpitations, a sense of anxiety, breathlessness, and orthopnœa.

Little and Callaway's case[15] well illustrates the extent to which such distension may reach.

A male imbecile aged thirty-four exhibited an inordinate appetite, but except for attacks of over-distension his health was good. On December 26, 1850, in addition to his meals, he devoured an unpealed orange and a pound of Banbury cakes. He was given aperients, which acted. He then became distended until he measured 45 inches in girth. His apex-beat was felt in the third interspace, and he suffered from orthopnœa. Calomel, opium, turpentine enemata, the rectal tube, and puncture, alike failed to open his bowels or relieve his distension. He died in seven days. The great gut was found to fill the abdomen. The transverse colon was 6 inches in diameter. The descending colon and sigmoid flexure together measured 20 by 12 inches, and were from $\frac{1}{3}$ to $\frac{1}{2}$ inch thick.

In a recent case which came under my notice, an ordinary attack of pneumonia terminated fatally in consequence of fæcal accumulations complicating it.

The vena cava and portal and spermatic veins may be pressed upon in exaggerated cases, and may lead to œdema of the legs and scrotum, varicocele, and hæmorrhoids.

In Peacock's[16] case, a man aged twenty-eight had suffered from constipation from his birth. He had been several times obstructed, and for eleven years before his death his bowels were relieved only by enemata. Ultimately he became obstructed and hugely distended, and all measures to relieve him failed. His legs, penis, scrotum, and the lower part of his abdomen, became œdematous, and he died.

At the post-mortem the whole of the colon was distended to a diameter of from 6 to 8 inches. Fifteen quarts of semi-solid fæcal matter was removed, and this mass had projected from the cæcum into the lower end of his ileum. Groups of stercoral ulcers were found in the lower part of the sigmoid flexure and in the middle of the transverse colon. The rectum was healthy.

Pressure may be exerted on the bladder and the uterus and its appendages, and the functions of these organs be interfered with. Finally, it has been stated that sciatica and neuralgia of the crural and other nerves may result from the pressure of fæcal masses.

Symptoms of Fæcal Accumulation.—A moderate degree of constipation is much more common in women than in men, but severe forms producing intestinal obstruction are more evenly distributed between the sexes.

Of the sixty-nine cases of intestinal obstruction due to fæcal accumulation admitted to the London Hospital in thirteen years, thirty-two were men and thirty-seven were women.

The history of such cases will reveal some of the causes already dealt with, such as errors in diet, sedentary and irregular habits, wasting illnesses, or the presence of neurasthenia.

It must be clearly stated at once that habitual constipation may be present, so that the bowels are as a rule emptied but once in three or even four days, and yet the patient may enjoy good health. In such cases the colon is not inflamed, nor are stercoral ulcers excited by the fæcal masses, and each act of defecation, when it does occur, clears the colon.

Leichtenstern has recorded some remarkable cases where patients with abnormally long colons defecated but once every few weeks, yet maintained good health.

As a rule, however, the subject of habitual constipation suffers from mild alimentary signs, such as a sense of fulness, tension, and discomfort, in the abdomen, from flatulence and belching of wind, and from a foul taste in the mouth.

The stools of such patients are lumpy, and are evacuated only by severe straining or by the free use of aperients. The fæcal masses are frequently covered by mucus. As the case progresses and fæcal matter loiters longer and longer in the colon, colitis and stercoral ulcers are produced. Attacks of irritative diarrhœa then punctuate the progress of the case, but only in a very partial manner clear away the accumulation which has excited them. Attacks of fæcal fever are frequently seen during the periods of constipation. The temperature falls as the bowel is temporarily cleared by a purge or an enema.

At a still more advanced stage of the disease the results of absorption from the colon become marked. The patient has a foul taste in his mouth, he loses his appetite, and his tongue is foul and his breath offensive. He grows thinner and his strength diminishes. His skin becomes dull and greasy. His conjunctivæ, eyelids, and axillæ, are often of a yellowish colour, and his sweat of an unwholesome odour.

In those whose nervous system is unstable, such as in the neurotic and the hypochondriacal, the brain is greatly affected by fæcal toxæmia. They become dull and lethargic or irritable and morose, or suffer from profound depression, to recover as soon as their lower bowel is evacuated.

In rare instances attacks of mania, insanity, or melancholia may be due to this cause in the predisposed. When constipation is far advanced, *fæcal tumours* are often found in the abdomen.

The masses are often most difficult to diagnose, and have been mistaken for sarcomata and carcinomata of the peritoneum, mesenteric glands, or omentum, and less often for neoplasms of the stomach, liver, spleen, kidneys, ovaries, and uterus. I have on several occasions seen masses of fæces produced on a milk diet mistaken for a fresh accumulation of pus in a pelvic appendix abscess which was being drained. On the other hand, an astonishing mass of fæcal matter may be effectually concealed in an abdomen, and yet produce urgent symptoms of its presence.

The most confusing cases are, however, those in which fæcal accumulation is associated with some other abdominal condition, such as appendicitis or tumours of the uterus and its appendages, or carcinoma of the colon. It is therefore of the greatest possible importance, to discuss how such masses may be recognized and distinguished. Careful and systematic examination of the abdomen, perhaps on several occasions or under an anæsthetic, will nearly always show that the tumour is in the course of the colon. The masses are peculiarly tender and sore. When large they are much more resonant on percussion than their size and prominence would lead one to expect. When they are of a putty-like consistency, a finger may be pressed into them so as to leave an impression, but it is seldom that this sign can be obtained. From some neoplasms they may be distinguished by the fact that their surface is always rounded and never flat.

Gersuny[17] has drawn attention to what he calls the "adhesion symptom." When steady pressure is made upon a fæcal tumour, and the pressure is slowly relaxed, the mucous membrane of the colon may be felt separating itself from the mass with a sticky sensation.

The most satisfactory proof of fæcal accumulation is to feel a large collection of hard scybala in the rectum. Unfortunately, this is far from a constant sign, for the masses may be in the sigmoid flexure, the cæcum, or the transverse colon.

A turpentine enema will nearly always bring away fragments of hard fæcal material, and it frequently happens that a series of enemata will produce increasingly large results. This is a very sure sign of a large accumulation in the colon.

Ultimately fæcal accumulation leads to attacks of colic, and these may terminate in acute ileus and death from paralysis of the gut. Such attacks are often precipitated by some purgative which has not acted. The patient is seized with most violent colicky pain in the abdomen, and becomes distended. The pain may be so violent as to produce syncope. Vomiting is usually present, but is seldom urgent or fæcal; the patient may become profoundly collapsed.

The case can usually be diagnosed by the previous history of chronic constipation, and of similar attacks of constipation, colic, vomiting, and distension, which were relieved by purgatives and enemata.

In very rare instances all the means adopted to empty the colon may prove of no avail, and the patient then dies with the symptoms of ileus and with profound collapse and vomiting.

Treatment of Fæcal Accumulation.—Only the main lines on which chronic constipation should be treated can be given here.

1. *Diet.*—The chief indication is to diminish proteids which leave little residue and are digested in the upper part of the alimentary canal, and to increase vegetables and raw fruit, which will occupy and stimulate the colon. Plenty of fluids should be taken, for constipation is sometimes due to a habit of drinking very little, but astringent red wines, tea, chocolate, and cocoa, should be avoided. A glass of lukewarm water night and morning is often beneficial. The food should be well masticated, and, if necessary, the teeth should be attended to.

Any idiosyncrasy of the patient must receive attention. Many foods act as a laxative with some, whilst they produce constipation in others. The most prominent example of this is milk.

2. *Muscular Exercises.*—Regular exercise should be insisted on, whether this takes the form of walking, horse-riding, cycling, lawn-tennis, or rowing. Where training in regularity and discipline are advisable, a progressive course of Swedish exercises, or gymnastics at a regular gymnasium, may be prescribed. Excess must be carefully avoided in this class of treatment, and the exercises must be persevered in for months or years.

3. *Massage* is adapted to those whose vigour and abdominal muscles are so greatly diminished that exercises are not possible. Massage should be carried out by one especially skilled in its application to constipation. The movements consist of tapping, punching, kneading, and rubbing, along the course of the colon. The object is to stimulate the movements of the colon, and not to force the contents along it. Whenever tenderness, diarrhœa,

pyrexia, and other absorption signs, indicate the existence of stercoral ulceration, massage must be practised with the greatest possible care. Machines have been invented to carry out these movements mechanically.

4. *Electricity.* — The Faradic current is employed. The abdominal wall is stimulated to contract, and it is said that peristaltic waves are excited in the intestines. In the abdominal method, both poles well wetted are passed deeply into the abdomen for short intervals. In other methods, one pole formed of a metal ball on an insulated stem is passed up the rectum. The other pole is then applied to the abdomen (recto-abdominal method) or spine (recto-spinal method).

5. *The Hydropathic Treatment* of constipation is carried out in special institutions. It consists of frictions, hot and cold douchings, sprays, and wet binders.

6. *Regularity.* — Whatever course of treatment is adopted, regularity of habits must be insisted on, especially in the matter of defecation. The patient must endeavour to defecate after breakfast every morning, even if he is often unsuccessful. He must even resist the desire to empty the bowel at other times. He must eat at regular hours, and devote due time and attention to the process. Constipation, like insomnia, is in a large degree a bad habit.

7. *Medicinal Treatment.*—We cannot here discuss the treatment of constipation by drugs, for which works on therapeutics should be consulted. As far as is possible, their use should be avoided and the above measures relied on.

Purgatives, such as calomel, castor-oil, colocynth, and jalap, should only be used to empty the colon at intervals. Laxatives, such as cascara, rhubarb, and aloes, may be used to increase the fluid poured into the large intestine.

Moderate doses of saline waters—Marienbad, Carlsbad, Homburg, Kissingen—taken on an empty stomach before breakfast are often effectual in clearing the bowel but their effect is not curative, the dose tends to constantly increase, and the constipation is confirmed.

The Treatment of Fæcal Colic and Obstruction.—As soon as it is perfectly clear that the vomiting, distension, pain, and constipation, are due to fæcal accumulation, opium may be administered in moderate doses to relieve the spasm and pain. Some recommend the administration of heroic doses of such purgatives as castor-oil, calomel, or Epsom salts, but it seems unwise to further distend an overburdened colon, or to flog into action a muscular wall incapable of propelling its contents.

The obstacle should be removed from below by means of repeated enemata, assisted if necessary by gentle abdominal massage. The buttocks should be raised, and a copious enema of 2 pints or more introduced slowly by means of a funnel and soft rectal tube. In soft collections, even when very large, warm soap and water is sufficient for the purpose. Where the mass is more resistant or the colon is very sluggish, turpentine may be added in the proportion of ½ ounce to 2 ounces to a pint of soap and water, but if this enema is repeated frequently the colon becomes acutely inflamed. Brine enemata (1 in 10) have a powerful stimulating effect on the muscular wall of the bowel. Castor-oil, Epsom salts, and asafœtida, have also been added to enemata.

When the above simple measures have failed to produce more than small and unsatisfactory results, a pint or more of olive-oil or an ounce or two of glycerine, may be introduced high up into the colon, and be left there as long as possible. A strong turpentine enema is then administered, and a very satisfactory result is usually obtained. In very obstinate cases the method of lavage may be adopted. A gallon or more of normal saline solution or hot water should be prepared at a temperature of 100° F. The patient should lie upon the left side, with the buttocks well raised upon a pillow. A biway rectal tube is introduced as high up the colon as possible. The irrigator should not be raised higher than 2 or 3 feet. The stream of water is then directed as far as possible upon the fæcal mass. This manœuvre may be repeated daily until the colon is completely emptied.

Where a very hard mass of fæcal matter occupies a more or less ulcerated rectum, it may be advisable to administer an anæsthetic, dilate the anus, and break up the mass with the finger or with instruments, and wash away the fragments.

The Surgical Treatment of Chronic Habitual Constipation.—From the above description of the organic changes which occur in a colon the seat of habitual constipation, it is evident that in the latter stages of the disease there is little hope of that organ returning to its normal structure and functions, or of the patient regaining even a moderate degree of health and well-being.

It is under these conditions that surgery has stepped in to relieve a condition which until recently was regarded as entirely in the domain of medicine.

The operation of entero-anastomosis, in which a portion of the intestine is excluded from the direct intestinal channel, has been employed in surgery for a long time. The special application of this method to the colon is known as "ileo-colostomy."

The ileum is divided at some distance from the ileo-cæcal valve, and the ends are invaginated by purse-string sutures. The proximal end of the ileum is then united by lateral anastomosis to the colon at some point below the obstruction. If the continuity of the ileum is not interrupted, the artificial opening tends to close. This operation has hitherto been employed for the most part in the treatment of carcinomata of the upper part of the colon.

Mr. Mansell Moullin[18] in October, 1900, was apparently the first to perform ileo-colostomy for chronic constipation. His patient was a woman who for seven years had suffered from atony of the colon and obstinate constipation which followed an attack of acute colitis. He did not divide the ileum. The patient was cured of her constipation.

Mr. Arbuthnot Lane[19] was, however, the first to definitely propose to treat chronic intractable constipation by exclusion of the colon from the alimentary canal. His argument is that, when the colon has been so misused that there is no hope of its recovery by medical means, and organic changes in its structure are already established, the patient is better without the use of this diseased organ. The operation is a simple one, the mortality is low, and the immediate benefit is great ; but neither Mr. Lane nor any other surgeon has as yet published a series of cases treated by this method giving the mortality and the ultimate results.*

Mr. Mansell Moullin's patient was still relieved from her constipation after four years. On the other hand, Sir William Macewen found that a patient of his from whom he had removed the termination of the ileum, the cæcum, and part of the ascending colon, suffered from irregular and unexplained attacks of diarrhœa, required to be carefully dieted, and never regained his normal health. We have seen that the ileo-colic sphincter regulates the flow of acid irritating chyme into the cæcum, which is specially adapted to deal with this fluid. When, however, the ileo-colic sphincter is excluded from the alimentary canal, and the ileum pours its contents uncontrolled into the rectum, it would seem probable that the latter would become ulcerated and inflamed. Several cases of peptic ulcer of the jejunum have been recorded as a result of a similar exclusion of the pyloric sphincter. These peptic ulcers are, however, so rare as to form no practical objection to gastro-jejunostomy, and, similarly, proctitis and diarrhœa do not appear to follow ileo-colostomy at all frequently.

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* This was written in 1907.—Ed.

A much more serious result from exclusion of the colon is the accumulation in the blind end of inspissated mucus and epithelial debris, which may form large masses. This difficulty has been recognized for many years in connection with colotomy. Mr. Harrison Cripps,[20] in a recent discussion of colotomy, speaking from an experience of more than 600 cases, said that in 20 to 30 per cent. there was trouble from decomposing mucous secretion in the distal part of the gut. In colotomy the difficulty can be met by syringeing through, but this treatment would not be available in ileo-colostomy unless an appendicostomy were performed for that purpose. In cases of my own I have prescribed a large, high soap-and-water enema to be given once a month, to obviate, if possible, this complication.

Mr. Lane has apparently also encountered this difficulty, for he has recently proposed to excise the colon in whole or in part at a subsequent operation.

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(iv.) **Gall-Stone Intestinal Obstruction**—*Definition.*—Gall-stone intestinal obstruction is said to occur when a large gall-stone becomes impacted in the alimentary canal.

Frequency.—This is a rare form of intestinal obstruction. At Leeds Infirmary only one case occurred in ten years,[1] at St. George's Hospital but one case in fifteen years.[2] The London Hospital records for the thirteen years 1893-1905 show that there were 669 cases of intestinal obstruction, including all varieties. Of these fifteen were due to gall-stones, a proportion of 1 to 44·6. These were a consecutive series, and probably more nearly repre-

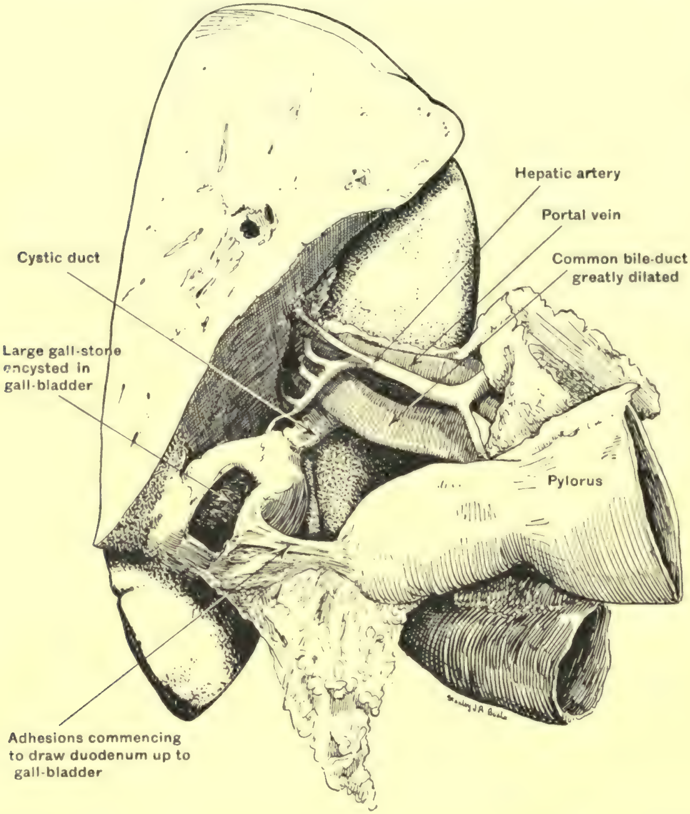
Hepatic artery
Portal vein
Cystic duct
Common bile-duct greatly dilated
Large gall-stone encysted in gall-bladder
Pylorus
Adhesions commencing to draw duodenum up to gall-bladder

Fig. 1.—From a Specimen in the London Hospital Museum, showing a Large Gall-Stone encysted in the Gall-Bladder.

Adhesions are commencing to draw the first part of the duodenum into contact with it.

sent the true proportion than the following series of cases collected from the literature:

Fitz states the proportion to be 1 in 13—namely, 23 cases due to gall-stones in 295 cases of intestinal obstruction. Gibson,[3] 1 in 17, or 40 cases out of 696. Leichtenstern, 1 in 28, or 41 cases out of 1,152.

The Previous History of Biliary Trouble.—A gall-stone, to obstruct the intestine, must as a rule be more than an inch in diameter.

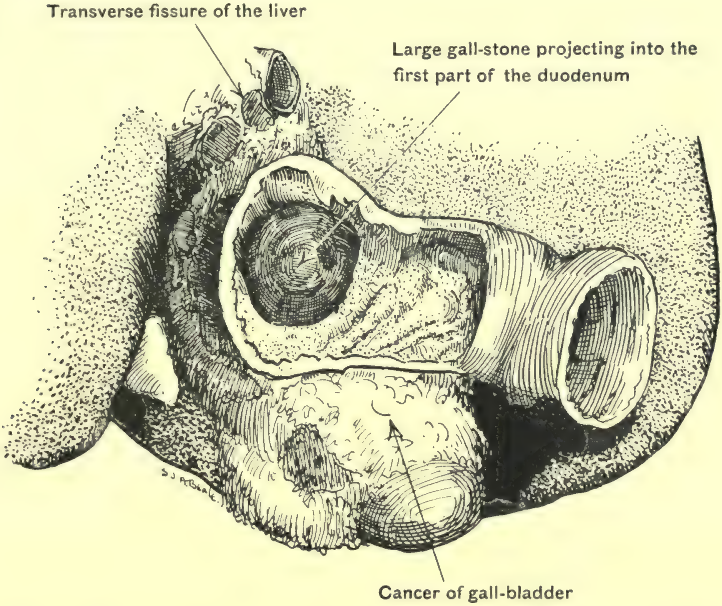
Transverse fissure of the liver
Large gall-stone projecting into the first part of the duodenum
Cancer of gall-bladder
S J A Beale

FIG. 2.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM, SHOWING A LARGE GALL-STONE PROJECTING THROUGH A FISTULOUS OPENING INTO THE FIRST PART OF THE DUODENUM.

Such large gall-stones are often single, and from their size cannot enter the biliary ducts. It is seldom, therefore, that a history of biliary colic and jaundice are obtained, although it does sometimes occur that smaller stones are associated with one or more large ones, and by entering the ducts produce both colic and jaundice.

As a rule the history is one of long-continued dull pain, with exacerbations at times in the epigastrium and right hypochondrium, which has been regarded as dyspepsia.

This pain is due to the chronic peritonitis and inflammation around the gall-bladder, which fixes it to the duodenum, and afterwards leads to the formation of a fistulous opening between the two.

Of the fifteen cases at the London Hospital, the previous biliary trouble was specially noted in nine. In one there had been no trouble of any sort. In two biliary colic and jaundice occurred. In two there was a history of violent spasms with vomiting. In four the pain was dull, and was regarded as dyspeptic.

The Morbid Anatomy and Pathology—1. *The Method of Entrance of the Calculus into the Intestine.*—Courvoisier[4] gives the following results of thirty-six autopsies. The stone had passed

Through a gall-bladder duodenal fistula twenty-five times.
" " ileum fistula once.
" " colon fistula once.
" " duodenal and colon fistula twice.
Through the common bile-duct seven times.

In three of these last seven cases it is only stated that the common bile-duct was large, and it is not definitely stated that no fistula existed. The remaining four are, however, indubitable cases. The gall-bladder was free from adhesions to the gut, and in one case the impression of two large stones which had obstructed the gut was found in the lower part of the common bile-duct. In two of these cases the common bile-duct was as large as the gall-bladder, and formed a continuous cavity with it in one of these. In two cases it is clear the gall-stone which obstructed the intestine was formed in the common duct. The stone almost invariably passes through a fistulous opening from the posterior surface of the neck, body, or fundus, of the gall-bladder into the first part of the duodenum. The encysted stone first leads to adhesive peritonitis around the gall-bladder, and binds it to the duodenum (*vide* Fig. 1). The wall between the cavities is then thinned away by the inflammation until they actually communicate (*vide* Fig. 2). For a greater or less time the end of the stone projects into the duodenum, and becomes white in colour as a consequence of continuous washing with acid chyme and the deposition of phosphates on it. This white nipple-like end of the stone is well shown in Fig. 4.

The final displacement of the stone has followed in several cases the administration of a purge.

In Case III. a smaller calculus passed down the cystic duct, and became impacted in the common duct (*vide* Fig. 7), producing biliary colic and jaundice. The bile stream was thus diverted up the cystic duct, and loosened the large encysted calculus, which passed through the fistula with the bile into the duodenum. In a few instances the fistula forms between the gall-bladder and the transverse colon. Such stones are voided spontaneously in the vast majority of cases, and account for those large rough stones of more than an inch in diameter which are evacuated by patients without severe signs of obstruction, and where it is almost incredible that they can have passed the ileo-cæcal valve. In rare instances it is said they may lead to obstruction in the sigmoid flexure or at the anus.

Where a stricture of the colon exists, even comparatively small gall-stones, which have passed down the common bile-duct, may become impacted, and lead to acute intestinal obstruction in just such a way as fruit-stones and other foreign bodies do. Mayo Robson[5] recorded a case in which a gall-stone was found in a pouch between two tubercular strictures of the ileum. In another case a gall-stone was impacted at the point where the small intestine passed below an omental band (Garrett).

In still rarer instances the calculus has ulcerated into a coil of jejunum.

2. *The Residence of Gall-Stones in the Intestine for Years before producing Obstruction.*—A few cases have been recorded in which it is claimed that the gall-stone entered the bowel fifteen years before it produced acute obstruction. Such gall-stones are presumed to be encysted in a pouch, and whilst they remain latent they become white in colour and covered with thick phosphatic accretions.

In Sir Thomas Smith's[6] case the stone was thought to have entered the bowel fifteen years before acute obstruction was produced.

In Mr. F. S. Eve's[7] case (*vide* Case VIII.) the calculus possibly remained in the bowel ten years before it became displaced, and passed down the intestine to lodge in the lower ileum, from which it was successfully removed. This stone was a dirty white colour, and its surface, which was washed and worn, tuberculated and friable, was largely formed of phosphates.

On the other hand, an ordinary small gall-stone passed by the common bile-duct may form the nucleus of a large intestinal accretion, as in a case recorded by Sir Frederick Treves.[8] He removed a calculus, measuring 1½ inches in its long axis, from the ileum of an old lady who for years had taken carbonate of magnesia daily. The nucleus of this calculus was formed by a small gall-stone, and its successive layers by magnesia and fæcal matter.

The presence of gall-stones which remain in the bowel for years is said to be indicated by evanescent attacks of colicky pain, vomiting, constipation, and sometimes distension.

The number of such cases recorded is, however, very few, and the evidence of the residence of the stone in the gut is not very conclusive. The attacks of colic, vomiting, and constipation, may well have been the result of localized attacks of peritonitis due to the encysted stone interfering with the duodenum and transverse colon.

3. *The Spontaneous Passage of Large Gall-Stones.*—H. D. Rolleston[2] states that 50 per cent. of cases of gall-stone intestinal obstruction are spontaneously relieved, sometimes quite suddenly; but the patient may not survive although she passes the gall-stones. This is shown by Case XIV., where, after being obstructed for a fortnight, and reduced to a parlous condition of collapse and fæcal vomiting, the patient passed three large gall-stones just under an inch in diameter, but nevertheless sank and died ten days later.

MacLagan[9] has also recorded a case in which a woman suffered four severe attacks of intestinal obstruction. She then passed spontaneously four large gall-stones, each 1 inch in diameter; but she died, and at the post-mortem only fringes of the ileo-cæcal valve remained.

When gall-stones larger than an inch in diameter are passed spontaneously without prolonged and severe symptoms of intestinal obstruction, it is practically certain that the fistula from the gall-bladder opened into the transverse colon, and that the calculus did not traverse the small intestine. The following is, apparently, of this kind:

For the history of the case I am indebted to Dr. C. P. Bremner, of Clapton.

Mrs. G., aged seventy-two, applied for relief from diarrhœa, and of "something in the back-passage which would not come away." For four years previously she had suffered from severe attacks of what she thought was "colic and spasms," but she was never confined to her bed and had never been jaundiced. Her present attack of diarrhœa had lasted fourteen days, and the sense of a foreign body in the rectum about the same time. A large gall-stone was removed by Dr. Bremner with the finger, and the patient was cured. The calculus measured 1 inch by 1¼ inches. Its surface was rough and black, and at its smaller end presented two concave facets. Its fractured surface revealed a lamellated structure.

The spontaneous passage of gall-stones may be greatly assisted by such drugs as opium, belladonna, and general anæsthetics, which relax the spasm of the gut. This matter will be referred to later under Treatment.

4. *The Site of Impaction.*—The point at which the stone becomes impacted is usually the lower part of the ileum.

Courvoisier[4] in fifty-three cases found the site of obstruction as follows:

In the sigmoid flexure in 2·4 per cent.
At the ileo-cæcal valve in 10 per cent.
In the ileum in 65·4 per cent.
In the duodenum in 21·4 per cent.

The small intestine becomes narrower from its upper to its lower end, and since small stones are more common than large ones, if they cause obstruction at all, it will be near the neck of this elongated funnel. In a former paper[10] the writer arranged in parallel columns a series of gall-stones according to their diameter and the point at which they had become impacted. This table showed that the diameter of the gall-stones bore a distinct relation to the distance they travelled down the alimentary canal before they became impacted. In other words, the larger the gall-stone causing the obstruction, the higher up it became impacted.

As a rule, any gall-stone less than 1 inch in diameter will pass spontaneously along the small intestine, unless it be unusually rough or angular.

The Pathological Basis of the Symptoms.—The first symptoms of gall-stone intestinal obstruction, severe pain and profuse vomiting, are due to an intense irritation of the duodenum by the calculus which has passed into it. The vomiting illustrates the general rule, that the higher up the intestines the obstruction is placed, the more profuse the vomiting will be. The vomiting in this form of obstruction is more profuse than in any other, and far exceeds the liquid matter swallowed. The dark colour of the early vomited matter is probably due to hæmorrhage from the edges of the fistula and from the raw interior of the gall-bladder, increased by the venous engorgement due to the vomiting.

In gall-stone intestinal obstruction there is no strangulation of the intestine. This is a pure obstruction of the lumen of the small intestine, without interference with its blood-supply and without paralysis of the nerves or muscles of the intestinal wall. Even this obstruction of the lumen is incomplete for the first few days, whilst the calculus is passing along the upper and wider portions of the small intestine, and during this period the constipation is partial and the pain colicky and intermittent.

When the calculus impacts, the obstruction becomes complete, the constipation absolute, and the pain continuous.

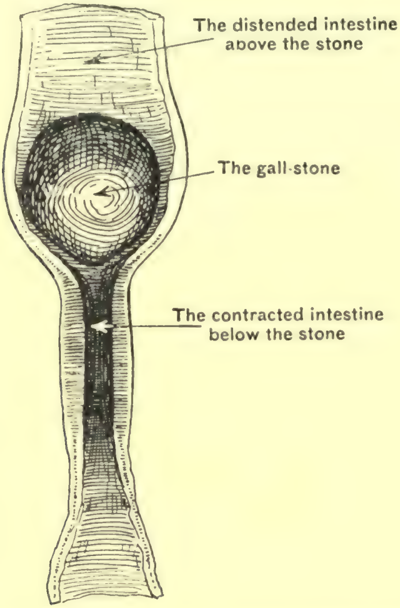
The distended intestine above the stone
The gall-stone
The contracted intestine below the stone

FIG. 3.—TO SHOW THE METHOD BY WHICH INTESTINAL OBSTRUCTION IS PRODUCED BY A GALL-STONE.

The Manner in which Obstruction is produced.—The obstruction is brought about mainly by the spasm of the intestine around and below the calculus, excited by the unusual contact of its rough surface (*vide* Fig. 3). Duplay and Reclus[11] emphasize this view. In the majority of recorded cases the operation has found the gut so contracted below and dilated above that the stone rested upon a septal-like projection in the gut, and could by no reasonable pressure be pushed on along the intestine. On the other hand, at many autopsies on these cases the calculus has been found lying quite loosely in the relaxed bowel, and it has been difficult to understand how it can have obstructed the gut at all.

Large doses of morphia and belladonna doubtless relax this spasm, and this will account for the success which has so often followed the exhibition of these drugs.

When the gall-stone has rested at one spot for some time, no doubt inflammatory swelling of the mucous membrane assists to make the obstruction complete.

The small intestine is, moreover, accustomed to propel fluid contents, and its interior is covered with valvulæ conniventes to delay the flow of the chyme along it. It has not a smooth interior and a powerful muscular wall like the rectum and sigmoid flexure, adapted to propel scybalous masses comparable to large gall-stones. The rough surface of the gall-stone is, moreover, ill-fitted to glide along the intestine, and should it remain there for any length of time, as it may have done in some cases already referred to, its bulk is further increased by rough intestinal accretions.

The Symptoms of Gall-Stone Intestinal Obstruction.—The writer has had the unusual fortune to encounter six of these cases in four years, and from a study and comparison of the symptoms of this series it has been possible to obtain a clinical picture by which these cases can be diagnosed with some degree of certainty.

The first case of this series of six was not suspected, but in every other case the diagnosis was made with varying degrees of probability.

The clinical picture is as follows: The patient gives in most instances a history of obscure epigastric or right hypochondriac pains for years, which are usually attributed to "dyspepsia." These pains must be attributed to the presence of a large encysted calculus, and to the adhesion of and subsequent ulceration of the gall-bladder into the first part of the duodenum.

Biliary colic and jaundice have occurred in only a minority of cases, for large stones are as a rule single. The patient is a fat woman about sixty years of age, and she is suffering from an acute obstruction of her small gut without distension. The intermittent and colicky character of the pain during the early days of the attack leads us to infer that strangulation of gut is absent, and that the obstruction is partial only.

Gall-stone intestinal obstruction occurs in women five times more frequently than in men. This is the same proportion of the sexes as is found in cases of gall-stones in the gall-bladder. (In the appended series of fifteen cases at the London Hospital, the proportion was 4 to 1.)

The average age of twenty cases collected by Mr. F. S. Eve[7] was 64. According to Sir Frederick Treves[8] the average age is 57 years. In fifty recent cases collected by Rolleston,[2] the average age was 62·7. In the London Hospital series of fifteen cases it was 59·3.

The onset of the attack is sudden. The pain is at first in the epigastrium, but when the stone has passed on into the jejunum and ileum it is referred to the umbilicus. The character of the pain is colicky and intermittent at first.

Vomiting is the great feature of the attack. It is severe, continuous, and profuse, and is early stained brown by bile or blood so as to closely simulate fæcal vomiting. In a case of Dr. Pye-Smith's, quoted by Sir Frederick Treves,[8] where the stone impacted in the upper jejunum, the patient vomited 1¼ gallons of bilious fluid in forty-eight hours, and died on the sixth day. The vomiting may, moreover, in many cases be divided into three stages (Barnard[10]). In the first it is sudden in onset, severe, continuous, and profuse in character and black in colour. In the second stage, when the stone has moved from a close proximity to the stomach, usually on the third day, the vomiting remits and becomes green; and as the bowels are often open on this day and the patient is not yet collapsed, she believes she is about to recover. In the third stage, when the stone has become impacted in the intestine, the vomiting returns, and is brown, like the fæcal stage of ordinary acute intestinal obstruction.

The constipation is the anomalous symptom. The patient has frequently precipitated the attack by taking a purge, and this purgative may act well during the first day or two of the disease. In the majority of cases fæces and flatus are passed during the first three or four days of the illness, in response to purgatives and enemata. Complete constipation is not present until the stone is finally impacted about the fourth or fifth day. Collapse is seldom well marked until the fourth day of the disease. The abdominal examination is as a rule negative. There may be a little tenderness on deep palpation in the gall-bladder region. The slight distension cannot as a rule be made out in such abdomens.

The stone can scarcely ever be palpated, but Mr. F. S. Eve[7] certainly felt one just above the ileo-cæcal valve, and the writer noted a lump in Douglas's pouch in a case in which the coil of ileum containing the calculus was found in that situation at the operation.

As will be seen from the distribution of impacted stones along the alimentary canal, the great majority will be placed in Douglas's pouch, dragged there by their weight, and might be felt by a rectal or vaginal examination.

Diagnosis.—The diagnosis is made on the unusual character of the above grouping of symptoms. An old fat woman with acute obstruction of the small intestine, yet of a partial character. The violence of the onset and of the vomiting, but the absence of total constipation. The shifting of the pain from the epigastrium to the umbilicus, and the remission of the vomiting on or about the third day. The history of long-continued and obscure pain in the right hypochondrium or epigastrium. The probability of the diagnosis will depend on the number of these groups of symptoms which can be elicited by a careful inquiry. Where they are all present, as in Case V., the diagnosis may be made with some confidence.

Duration and Prognosis.—Dr. Rolleston[2] states that 50 per cent. of cases not operated on are spontaneously relieved by the passage of the calculus. As has been already pointed out, all these cases do not survive. In unrelieved cases death occurs in from five to ten days. The mortality after operation is very heavy. Thirteen of the fifteen cases at the London Hospital were operated on, and of these eight died, a mortality of 61·6 per cent. This mortality is chiefly to be attributed to the great average age of these patients, to their degenerate tissues and organs, and to the fact that, owing to the remission of their symptoms on the third day, they are submitted to operation at a very late date.

Thus the average age of the ten cases that died at the London Hospital was sixty-three; of those that lived, but fifty-three. Those that died had been obstructed on the average just under eight days; those that lived, only four days. No case recovered which had been obstructed five days or more, and the only very old woman (aged seventy-three) who lived was thin.

Waring[12] quoted the following figures:

Lobstein,[13] 31 cases of operation; mortality, 61·3 per cent.
Courvoisier,[4] 125 cases of operation; mortality, 44 per cent.
Schuller,[14] 82 cases of operation; mortality, 56 per cent.
Jonathan Hutchinson, Senior;[15] mortality, 50 per cent.

Tillman[16] states that recent results are much more favourable, and he quotes Korte as having saved four out of five consecutive cases.

Treatment.—Every case in which acute intestinal obstruction is diagnosed should be operated on at once.

If gall-stone intestinal obstruction is suspected, early operation is all the more important for the reasons already stated.

When the abdomen has been opened, distended, or collapsed, gut will present according to the position of the stone. This form of obstruction is almost the only one in which a hand swept at a venture round the lower abdomen and pelvic pouch is almost certain to detect the cause of the obstruction at once. Should this measure fail, the gut must be systematically followed to the stone. When distended gut presents, it is better to follow the collapsed ileum from the ileo-cæcal valve, for the stone must be low down in the alimentary canal. When collapsed gut predominates in the field of operation, a distended coil should be drawn out and followed to the right, when the stone will be almost immediately encountered in the jejunum. The coil is drawn out, emptied, and a clamp applied. Sponges are packed around, and the stone delivered on to a gauze pad through a longitudinal incision, which is immediately closed by a double layer of sutures. The operation is exceedingly simple and rapid.

Some surgeons have recommended pushing the stone on through the ileo-cæcal valve, and leaving it to be spontaneously evacuated. This procedure is seldom possible, and with modern methods of intestinal suture the time so expended would be far more dangerous than the risk of peritoneal infection after incision and suture.

Others have recommended that the stone should be pushed up the intestine, so that the incision in the gut may not be through the area inflamed by the impacted stone. This procedure should be reserved for cases in which there is clear evidence of inflammation and ulceration ; in all others it is needless and wastes valuable time.

Where the patient will not submit to operation, or where skilled surgical assistance is not available, the patient should be placed deeply under the influence of opium and belladonna.

Mr. J. Hutchinson,[15] who is opposed to surgical interference in this disease, advocates in addition general anæsthetics and rectal injections of air and fluid.

Treves[8] quotes a case of Martin's[17] in which massage on the sixth day was followed on the seventh by the passage of one large and ten small gall-stones.

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Cases of Intestinal Obstruction due to Gall-Stones.

This series consists of a record of the six cases treated by the writer, with a summary of the eight further cases which were admitted to the London Hospital from 1893 to 1905.

These fourteen cases are all that occurred in the consecutive series of 669 cases of intestinal obstruction of all kinds treated during that period.

CASE I.—Intestinal obstruction in a woman aged thirty-seven, for four days, due to a gall-stone $1\frac{1}{8}$ inches in diameter, impacted in the ileum about 5 feet from the cæcum. Enterotomy; recovery.

R. F., a married woman aged thirty-seven years, was sent up to the London Hospital on January 13, 1901, by Dr. MacDonnell, of Stoke Newington, with a diagnosis of intestinal obstruction and the following history: Until nine months ago she had enjoyed very good health. She had never suffered from biliary colic or jaundice, nor had she any previous attack of intestinal obstruction. For nine months before admission to the hospital she had, however, suffered from an aching pain in the right side of the abdomen, which prevented her from lying on that side. This pain was at times so severe as to make her feel sick. On Wednesday, January 9, 1901, she was suddenly seized with a "griping" pain in the right side of the abdomen, below the ribs. On Thursday, January 10, 1901, she took a purge, and her bowels were opened several times on Friday, but the pain was not relieved. From this time she had absolute constipation; neither flatus nor fæces were passed, but she suffered from tenesmus. Vomiting commenced on Saturday, January 12, 1901. It was incessant, profuse, and from the very commencement brown in colour, and smelled of fæces. The patient and her husband volunteered the statement that "she was passing her motions from her mouth." She was in great pain and in a condition of severe prostration.

On admission to the hospital the abdomen was found to be flaccid, and neither distended nor tender on palpation. The pulse was 100 per minute and of fair volume and tension. The tongue was dry and covered with white fur. The patient complained of great thirst, and about an hour after admission vomited dark, feculent-smelling matter. *Per vaginam* a mass, which was taken to be fæcal, was felt in the situation of Douglas's pouch. On examining the rectum it was found to contain some fæces, and the same hard mass could be felt above and in front.

Treatment.—Three enemata, containing turpentine, were given at intervals of about a couple of hours. The first brought away a little fæcal matter. The other two were without any result either of fæces or flatus, and the hard mass could still be felt from the rectum. The vomiting was now more urgent and the patient's general condition had not improved.

Operation.—The abdomen was opened by the usual median laparotomy incision below the umbilicus. A coil of congested and distended small gut projected. The inner aspect of the hernial rings and the umbilicus were then explored with the hand and found to be normal. The cæcum and appendix were examined. The former was found empty and contracted. The empty coil of ileum entering it was drawn out of the wound and rapidly followed up. At a distance of about 5 feet from the ileocæcal valve the gut was found to contain an ovoid, hard mass, which was recognized as a gall-stone. The gut was empty and contracted to a very small lumen below the stone, whilst above it was greatly distended and congested. The sudden narrowing of the gut formed a kind of septum, on which the stone rested and blocked the narrow orifice left. The coil of gut containing the stone was delivered out of the wound and packed round with sponges. It was then emptied of fæcal matter and clamped above and below. A short incision was made in the length of the gut and the stone squeezed out. The incision was then closed by a continuous suture of No. 1 silk, through the peritoneum muscle and mucous membrane, as the mucous membrane was too thin to be sewn separately. The wound was invaginated by interrupted Lembert sutures. The gut was well washed out with hot saline solution and returned to the abdomen. The abdominal wound was closed with through and through silkworm-gut sutures without a drain.

Progress.—The bowels were opened on the second day by enema. The abdominal wound healed by first intention, and the patient made an uninterrupted recovery, which was, however, a little delayed by cystitis and some retention of urine.

The *calculus* (*vide* Fig. 4) was egg-shaped, and measured $2\frac{1}{2}$ inches in length, $1\frac{1}{8}$ inches in diameter, and $3\frac{1}{2}$ inches in circumference.

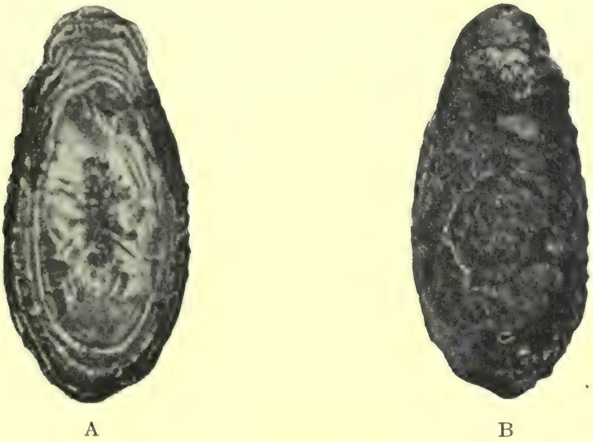

FIG. 4 —GALL-STONE REMOVED IN CASE I. FROM THE ILEUM.

A shows, on section, the white centre, end, and dark crust upon the body of the calculus. B shows the rough and dark-coloured exterior.

It weighed 294 grains when dry. The structure was lamellated, but the central part, of nearly pure cholesterin, had recrystallized into a stellate arrangement. The narrow end presented a nipple-like projection of pure white cholesterin, which passed at a neck into the general body of the calculus, which was covered with a layer of dark brown friable material. It will be seen in the diagram that the layers passed without interruption from the brown friable part to the pure white portion. It seems, therefore, probable that this nipple projected for a long time into the duodenum, and that the constant washing with acid chyme dissolved out the pigment from the exposed portion. Dr. Hunter, assistant-bacteriologist to the London Hospital, kindly examined a scraping taken from the centre of the stone, and found it to contain *Bacillus coli communis.* No typhoid bacilli were found; but in connection with the early age at which this woman developed a large gall-stone, it is interesting to note that the obscure hepatic signs of the stone followed an attack of typhoid some years before. Attention had been drawn to this point first by Bernheim in 1889, and afterwards by Dufort, Chiari, Mason, and Osler.

CASE II.—Intestinal obstruction in a woman, aged sixty-three, for five days, due to a calculus $\frac{7}{8}$ inch in diameter, impacted in the ileum about 5 feet above the cæcum. Enterotomy; death.

In June, 1901, Mrs. M. M., aged sixty-three years, was admitted under the care of my colleague, Dr. F. J. Smith, for intestinal obstruction, and I was asked by him to see her, with a view of operation.

History.—She gave a very clear history of previous biliary trouble. She had suffered for years from obscure pains in the right hypochondriac region, which she had always supposed originated from the liver, but she had never been jaundiced. While she was stopping at Hastings in the summer of 1900, she had a very severe attack of pain in the region of the liver, accompanied by continuous retching, but not by actual vomiting. Her doctor told her the attack was due to the passage of the gall-stone. No search was, apparently, made for this in the motions.

The *present attack* was apparently produced by a purgative. On Sunday night, June 10, 1901, the patient took a pill, and this acted very thoroughly on Monday. On Monday, June 11, 1901, at 11 p.m., and five days before she came to the hospital, she was seized with the most violent pains in her right hypochondrium, and with profuse and continuous bilious vomiting. She continued in this condition all night, but improved considerably in the morning (Tuesday, June 12, 1901). The sickness was much less. The colicky pains were, however, nearly as bad, but had now shifted to the umbilical region. An enema was administered, and produced a good result; her doctor was called in. On Wednesday, June 13, 1901, the patient was still retching, and the vomited matter was now brown and offensive. Her medical man wished her to come up to the hospital, but she refused all surgical treatment. Next day, Thursday, June 14, 1901, an enema produced a small result; she was much better; the vomiting almost ceased, and she obtained some sleep. On Friday, June 15, 1901, she was much worse. The vomiting was now distinctly fæcal. On Saturday morning, June 16, 1901, she consented to operation, and was brought up to the London Hospital.

Condition on Admission.—Her general health was very bad. There was bronchitis, and her pulse was rapid and feeble. The abdominal examination was mostly negative in result. There was no distension and very little tenderness. No tumour could be felt either by palpating the abdomen or on examining the rectum and vagina, although Douglas's pouch was most carefully examined. The hernial rings were empty. A diagnosis of acute intestinal obstruction by gall-stone was made on the following grounds:

1. The patient was a woman, and sixty-three years of age, suffering from acute intestinal obstruction.
2. The definite history of previous hepatic pain and vomiting.
3. The clear history that the pain in the present attack began in the hepatic region, and shifted to the umbilical region later.
4. The early profuse and bilious vomiting which remitted, and later returned and became fæcal.
5. The late and incomplete constipation.
6. The absence of distension, which pointed to an obstruction of the small intestine.

Operation.—Anæsthetic chloroform. During the whole operation she was regurgitating large quantities of fæcal vomitus. The usual laparotomy incision was made. As soon as the belly was opened, congested and dilated coils of small intestine presented, and some blood-stained fluid escaped from the peritoneal cavity. A hand swept round the pelvis proved the hernial rings were empty. The cæcum was then examined and found collapsed. The entering coil of collapsed ileum was brought out of the wound and followed up for about 5 feet, when a large calculus was found obstructing the lumen of the gut. The ileum above the stone was greatly dilated and congested; below it the gut was pale and contracted, so that the stone, as in the previous case, rested on a septum. The stone was removed, and the incision closed in an exactly similar way to that described in Case I. The contents of the intestine were heard gurgling past the obstruction before the belly was closed. A stomach-tube was passed, and several pints of feculent fluid were removed from the stomach, which was then washed out until the returning fluid was fairly clear. The patient's condition was, however, very bad after the operation. She was collapsed and had a continuous rattle in her throat.

Progress.—On the following morning she was cold, blue, and delirious, and her respiration was very rapid—48 to 52 per minute. Her temperature had risen to 100° F. She had not been sick since the operation, and she had profuse diarrhœa with incontinence all night. She died in the afternoon, twenty-four hours after the operation.

The *stone* was barrel-shaped and faceted at both ends. It was a dark coffee colour, and its outer layer was friable. The diameter was $\frac{7}{8}$ inch; circumference, 3 inches. The weight, when dry, was 103 grains. The faceting on either end appeared to indicate that this stone was one of three or more stones, of which one or more remained in the gall-bladder, whilst the passage of the other had caused the previous attack of pain and vomiting. The post-mortem examination, however, indicated that no large stone had previously passed, but that the upper facet was produced by several small stones found in the gall-bladder.

Post-Mortem.—The incision in the gut was soundly closed and surrounded by plastic peritonitis. There had been no leaking of intestinal contents. The predisposing causes of death were found in small, contracted, granular kidneys, the capsules of which would not strip, in a fat and flabby heart and an atheromatous aorta. The endocardium and endarterium were deeply stained with blood-pigments as in a septicæmia. The right lung was solid at its base and sank in water. The bronchial tubes contained purulent matter. The left lung was in much the same but a somewhat earlier condition. The liver, gall-bladder, duodenum, pancreas, and stomach, were removed *en masse* so as to be dissected and preserved.

The following structures were carefully dissected out (*vide* Fig. 5):

(1) The duodenum; (2) the gall-bladder and the calculi it contained; (3) the hepatic, cystic, and common bile ducts and the pancreatic duct; (4) the portal vein, hepatic artery, and vena cava: these latter structures were normal, and need not be again mentioned.

1. The *Duodenum* was hung up by adhesions to the neck of the gall-bladder. It passed from the pylorus 1 inch upward and to the right, and was adherent to the middle of the left border of the gall-bladder. It then passed in a loop or festoon behind the gall-bladder, and was again adherent to the right side of the neck of that viscus. It was here that the fistula bimucosa existed. I was able to work the calculus, which had been taken from the ileum, back up the duodenum and through this fistula until it occupied its old quarters in the gall-bladder. A diverticulum was found springing from the lower border of the duodenum immediately opposite the fistula bimucosa. This diverticulum was buried in the head of the pancreas, and was the size of a large hazelnut. At first it was thought that the stone which had caused intestinal obstruction had lain for some time in this diverticulum after leaving the gall-bladder, but on actual trial the diverticulum was found to be far too small to hold the stone. It is possible that this diverticulum served as a by-pass to permit the flow of chyme past the obstruction caused by the gall-stone projecting through the wall of the duodenum immediately opposite to it. The entrance of the common bile-ducts into the duodenum was normal in position and structure. The third and fourth parts of the duodenum showed no pathological changes.

2. *The Gall-Bladder and Calculi.*—The gall-bladder was found divided into four loculi or compartments, separated from one another by dense fibrous septa, and communicating by orifices of not more than $\frac{1}{4}$ inch in diameter.

(1) The first, at the fundus, contained twelve small calculi, the smallest of which was the size of a tare, and the largest that of a pea.

(2) The second compartment, about the middle of the gall-bladder, contained a solitary large smooth calculus faceted at both ends, and only a little smaller than that taken from the ileum. It weighed 97 grains when dry.

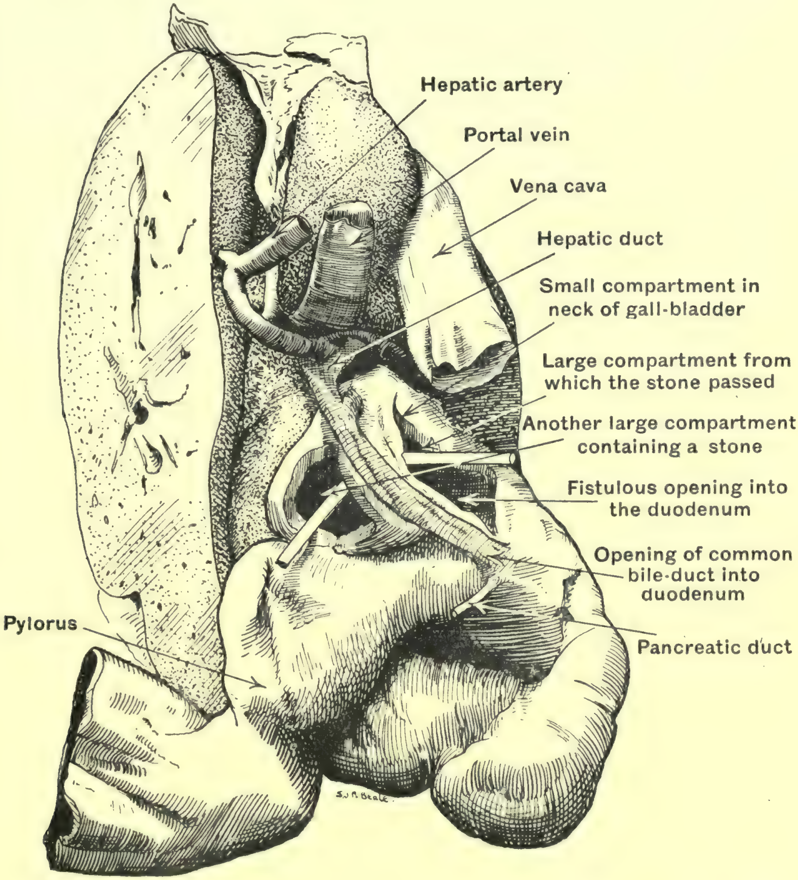
Hepatic artery
Portal vein
Vena cava
Hepatic duct
Small compartment in neck of gall-bladder
Large compartment from which the stone passed
Another large compartment containing a stone
Fistulous opening into the duodenum
Opening of common bile-duct into duodenum
Pylorus
Pancreatic duct
S. J. A. Beale

FIG. 5.—SHOWING THE FISTULOUS OPENING THROUGH WHICH THE GALL-STONE PASSED INTO THE DUODENUM.

Three of the four compartments into which the gall-bladder was divided are also shown. The fourth was in the fundus and is concealed by the duodenum. This specimen is in the London Hospital Museum.

(3) The third loculus was near the neck of the gall-bladder, and it was this one which communicated with the duodenum. It was for the most part empty, so that the calculus taken from the intestine was reintroduced with ease, but in one corner a small calculus the size of a pea was found.

(4) The fourth and last compartment was at the very entrance to the cystic duct. It was not larger than a pea, and communicated by only a pinhole with the third loculus. It was completely filled by a round tuberculated and pale-coloured calculus. The cystic duct had apparently been obliterated by this stone, for no probe could be passed either from this compartment into the common duct or in the reverse direction. Perhaps this stone was impacted in the cystic duct, and had led by its complete obliteration to the necessity of a fistula into the duodenum.

3. *The Hepatic, Cystic, and Common Ducts.*—The hepatic and common bile ducts were very little, if at all, dilated. The common duct at its widest point would have just taken a medium-sized pencil. Its orifice into the duodenum was not dilated. The cystic duct, as has already been said, was obliterated, and existed only as a fibrous cord connecting the common bile-duct with the neck of the gall-bladder. The common bile-duct was clearly demonstrated to its entrance into the duodenum, and certainly the large stone removed from the ileum did not pass this way. The pancreatic duct was in no way abnormal.

CASE III.—Intestinal obstruction in a woman, aged sixty-eight, for five days, due to a gall-stone $1\frac{1}{4}$ inches in diameter, impacted in the jejunum 5 feet below the duodenum. Enterotomy; death.

I. P., a German woman aged sixty-eight years, was admitted on the afternoon of September 11, 1901. She was in so collapsed a condition and spoke so little English that no reliable history could be obtained. Apparently her illness began two months before, with severe abdominal pain and jaundice. Five days before admission she became acutely ill with profuse bilious vomiting and severe pain in the belly; but I could not discover where the pain had been. For two days she had passed no motion. The patient was an enormously fat woman. She was collapsed and nearly pulseless; when admitted her pulse-rate was 103, but very feeble; her temperature was 95° F. An enema produced a small result. Her belly was soft and voluminous, and not distended or tender; no tumour could be felt, and the hernial rings were all clear. The rectal examination was omitted. Under stimulants and a coffee-and-brandy enema she rallied a little in four hours, and it was then decided to operate, as a diagnosis of gall-stone intestinal obstruction had been made. The operation was carried out under eucaine local anæsthesia, and she experienced no pain except at the skin incision and when the mesentery was pulled upon. The usual laparotomy incision from umbilicus to pubes was employed, and 3 inches of fat were cut through before the muscular wall was reached. When the peritoneum was opened, most of the small gut was found collapsed and pale, but one coil in the upper part was congested and greatly distended. This coil was followed down, and almost immediately a large gall-stone in the gut was drawn into the wound, surrounded by sponges, cut on in the line of the gut, and evacuated. The gall-stone rested on a septum formed by the junction of the distended and collapsed intestine, similar to that described in Cases I. and II. In order to give her the best possible chance of recovery, it was determined to drain the gut at the point of suture, and accordingly a Paul's tube was tied in and fixed in the upper angle of the wound, and the remainder sutured with silkworm gut.

Progress.—Her condition was improved after the operation. She did not vomit, and her lungs remained clear. She evacuated fluid fæces freely from the tube, and, stimulated with strychnine and coffee-and-brandy enemas, she lived forty-eight hours.

Post-Mortem.—Only a partial autopsy was allowed. The liver and intestines were removed. The point of obstruction was found to be 5 feet from the duodeno-jejunal flexure, and 10 feet from the ileo-cæcal valve.

The *calculus* (*vide* Fig. 6) was large, rough, black, and barrel-shaped. It measured 1¾ inches in length, 1¼ inches in diameter, and 3¾ inches in circumference, and weighed, when dry, 234 grains. At its lower end was a smooth white area similar to that on the end of the calculus in Case I., which I presume had projected into the duodenum for some time. On section it was found to be very friable, amorphous in structure, and not lamellated. The structure was coarse, and innumerable little crystals sparkled on its surface.

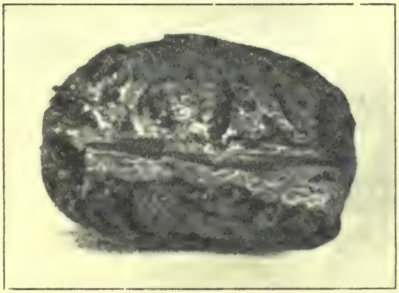

FIG. 6.—CALCULUS REMOVED FROM CASE III.

I recently opened a gall-bladder soon after an attack of cholecystitis, and found it filled with gall-stone mud, in which were the crumbling remains of a number of small faceted gall-stones. It appeared, indeed, that the acuteness of the inflammation had broken up a multitude of faceted gall-stones into this mud. If the moisture were absorbed from this mass, and it consolidated with the formation of minute crystals of cholesterin, it would produce just such a large friable, amorphous calculus as in this case. I must admit, however, that I found no trace of partially eroded calculi on section; and of course this theory of the origin of large gall-stones would not apply to those which are lamellated.

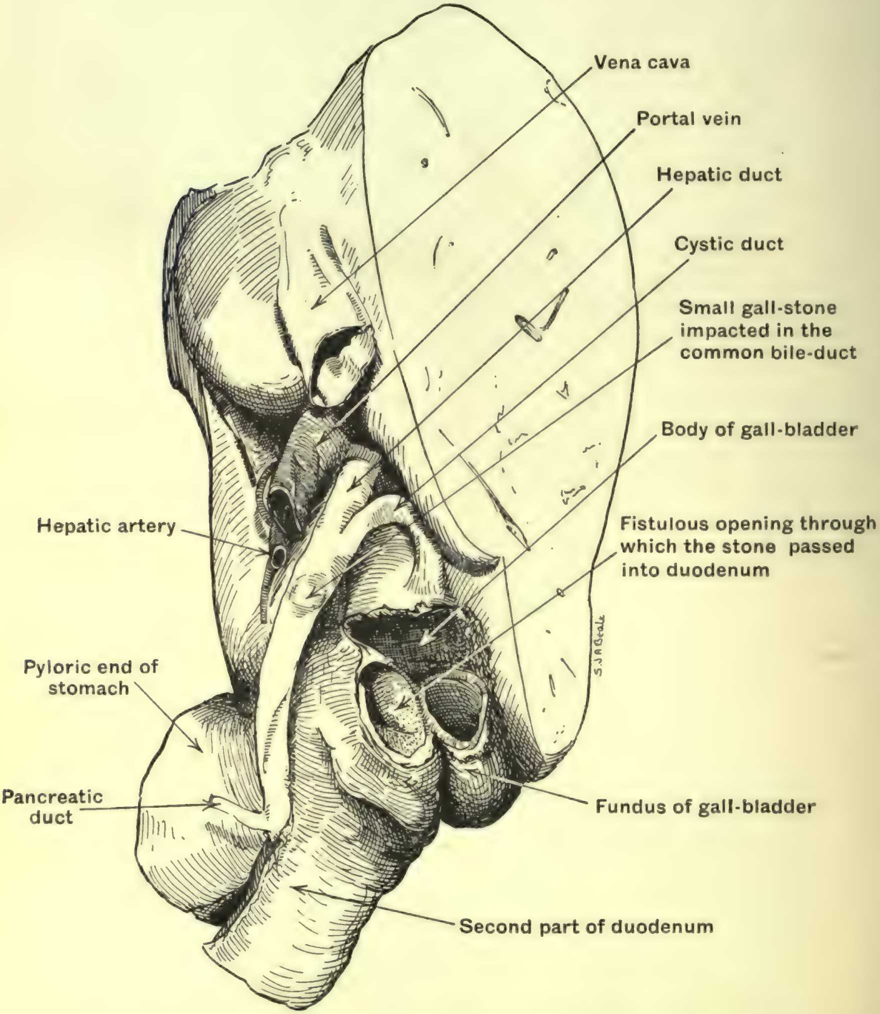

FIG. 7.—SHOWING FISTULOUS OPENING FROM THE GALL-BLADDER INTO THE DUODENUM, THROUGH WHICH THE STONE PASSED.

Another stone is seen impacted in the common bile-duct. This specimen is in the London Hospital Museum.

The liver, gall-bladder, bile-ducts, and duodenum, were dissected as in Case II., and this specimen was shown with the other at the meeting of the Pathological Society (*vide* Fig. 7).

The *duodenum* was curiously twisted, and indeed inverted. The stomach, pylorus, and first part of the duodenum, had apparently been forced downward, so that the second part ran up instead of down, and the duodeno-jejunal flexure and its suspensory ligament (muscle of Treitz) were on a much higher level than the pylorus and first part of the duodenum (Fig. 8). This was probably produced by tight-lacing in youth, and the formation of the gall-stones may have been due to this cause, as has been urged by

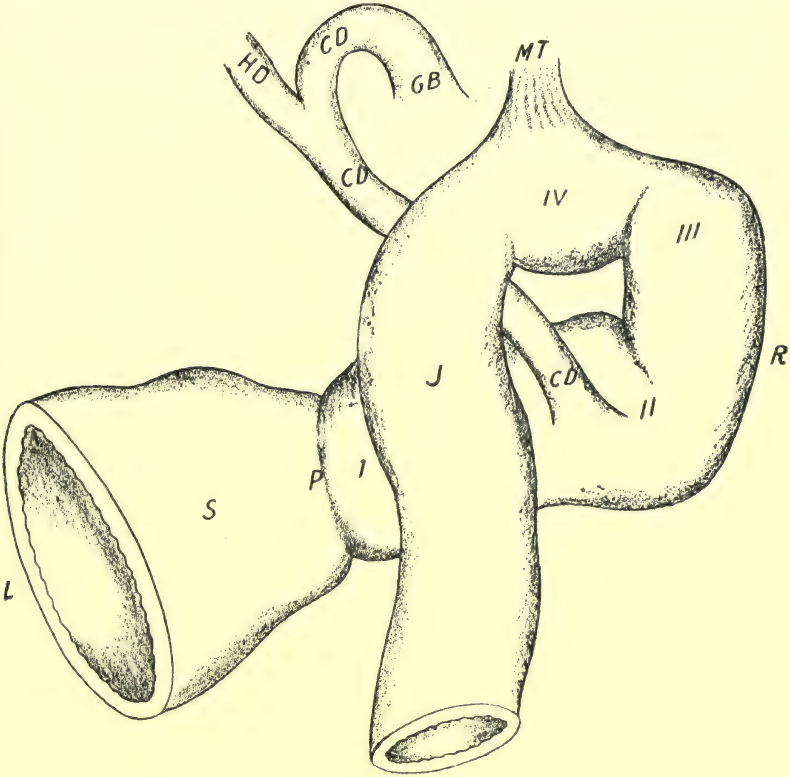

FIG. 8.—TRUE POSITION OF DUODENUM: POSTERIOR VIEW.

S, Stomach; P, pylorus; I, II, III, IV, parts of duodenum (it is to be noted that the second and third parts both ascend to the junction with the jejunum, held up by MT, the muscle of Treitz); GB, gall-bladder; CD, cystic duct; HD, hepatic duct; CD, common bile-duct.

Professor Arthur Keith, who regards gall-stones as a part of what he has called "corset disease." The first part of the duodenum formed practically the major part of the posterior wall of the gall-bladder, and here a large ragged fistula existed between them.

The *gall-bladder* was not greatly enlarged, but its walls were thick, ulcerated, and adherent to the duodenum behind as described. It passed by a funnel-shaped neck into a dilated *cystic duct*, and this joined an equally dilated *hepatic duct.*

The *common duct* when flattened out was $\frac{1}{2}$ inch in diameter and somewhat thickened. About an inch below its origin a small elongated stone, the size of a small bean, was impacted. No doubt it was the passage of this stone which caused the onset of the symptoms two months before death with jaundice. When this jaundice subsided (for the patient was not more than tinged when admitted to the hospital), it was probably owing to the escape of bile by way of the gall-bladder around the calculus and through the fistula. It also seems probable that this stream of bile ultimately loosened the stone and led to its expulsion into the duodenum. The *pancreatic duct* was not dilated. The *pancreas* was firm, and appeared somewhat fibrous, but it had been preserved in formalin.

CASE IV.—Intestinal obstruction in a woman, aged sixty-eight, for five days, due to a gall-stone $\frac{15}{16}$ inch in diameter, impacted 8 feet above ileo-cæcal valve. Enterotomy; death from shock.

Ann Gibson, a stout married woman aged sixty-eight, was admitted to the London Hospital on May 31, 1902. She had suffered from no previous abdominal trouble of any sort. She had been constipated during four days, when, five days before admission, she was seized with severe abdominal pain, followed shortly by profuse vomiting. Her bowels were opened by enemata on the second and fourth day of her illness. On the third day of the attack she felt much better, and the vomiting remitted.

On admission her condition was fair. Her temperature was, however, subnormal (97° F.), but her pulse was but 80 beats per minute and full. Her tongue was furred and dry. Vaginal, rectal, and abdominal examination were negative except for a little tenderness in the right hypochondrium. Two turpentine enemata were given, and produced no result in fæces or flatus. The patient soon after admission vomited some brown, fæcal-smelling fluid.

Operation.—The abdomen was opened in the middle line below the umbilicus. Collapsed and distended coils of small gut presented. A hand introduced encountered a round stone about the size of a walnut in the upper ileum. The coil was drawn out, emptied, clamped, and packed round with sponges. The stone was evacuated through a longitudinal incision, and the aperture closed at once by a double layer of No. 1 silk sutures. The gut was washed with saline and reduced. The abdominal wall was closed with through and through silkworm-gut sutures. At the close of the operation she was not collapsed, and on her return to the wards was no worse than before the operation. Her condition was so good that stimulants and transfusion were omitted. She collapsed suddenly early next morning, and died in an hour.

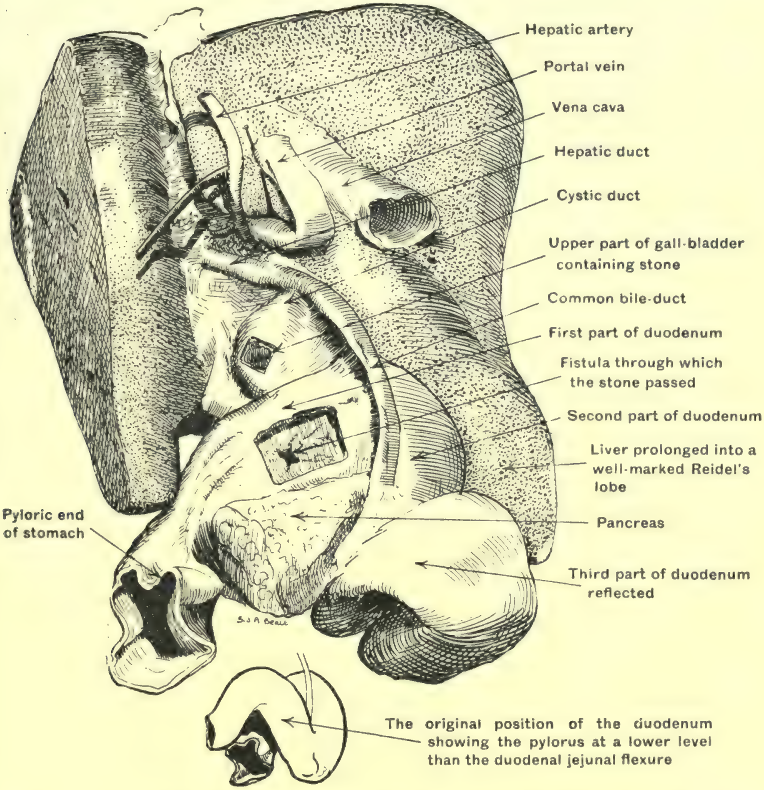
Hepatic artery
Portal vein
Vena cava
Hepatic duct
Cystic duct
Upper part of gall-bladder containing stone
Common bile-duct
First part of duodenum
Fistula through which the stone passed
Second part of duodenum
Liver prolonged into a well-marked Reidel's lobe
Pancreas
Third part of duodenum reflected
Pyloric end of stomach
S.J.A. Beale
The original position of the duodenum showing the pylorus at a lower level than the duodenal jejunal flexure

Fig. 9.—Showing the Fistulous Opening between the Gall-Bladder and the First Part of the Duodenum, through which the Stone passed.

This specimen is in the London Hospital Museum. The small diagram shows the enteroptosis of the pylorus and first part of the duodenum.

This case impressed on me the importance of stimulating all such abdominal cases after operation, even when their general condition is good, and my surgical Sister reports that their progress has been much more satisfactory since this was adopted as a routine. As a rule hypodermics of strychnine ($\frac{1}{60}$ grain) and caffeine (2 grains) are given alternately every two hours. Where shock is present, 4 to 6 pints of saline are transfused continuously beneath the skin on the outer aspects of the thigh.

At the autopsy the stone was found to have been impacted 8 feet above the ileo-cæcal valve and 9 feet below the duodenum. The suture line had not leaked, and there was no peritonitis. The usual condition of general fatty infiltration was found in all her organs.

The liver and duodenum were removed for careful dissection, and are shown in Fig. 9. The stone was almost circular. It measured 1 inch in length, and $\frac{15}{16}$ inch in diameter. Its surface was brown and rough.

Dissection of Liver and Gall-Bladder and Duodenum.—The right lobe of the liver was prolonged into a long Riedel's lobe, which showed at the right margin an indentation. From its general shape, there was little doubt that it was the product of tight-lacing. The gall-bladder was displaced into the median notch.

The first part of the duodenum crossed the body of the gall-bladder transversely, and over this area they were firmly adherent. In the centre of this area of adhesions a fistulous opening connected the two cavities, distant 1 inch from the pylorus.

Another gall-stone, brown and tuberculated, and the size of a hazelnut, was encysted in the neck of the gall-bladder and plugged the cystic duct. The cystic duct was but $\frac{1}{4}$ inch long. The hepatic and common duct appeared healthy, but of somewhat larger lumen than normal.

The duodenum was inverted, as described in Case III., Fig. 7, so that the pylorus and first part of the duodenum were on a lower level than the duodeno-jejunal flexure. No doubt this enteroptosis was also a result of tight-lacing.

CASE V.—Intestinal obstruction in a woman, aged forty-nine, for three days, due to a gall-stone $1\frac{3}{16}$ inches in diameter, impacted in the lower ileum. Enterotomy; recovery.

Annie Askew, a stout married woman aged forty-nine, was admitted to the London Hospital on September 13, 1902. She had never had jaundice or biliary colic, but for years had suffered from dyspeptic pains in the right hypochondriac and epigastric regions.

Two years previously she had an attack of peritonitis in the right iliac region and lower abdomen, accompanied by a foul vaginal discharge. She had been quite well recently. The present illness commenced suddenly three days before admission, with severe pain across the belly above the umbilicus. Vomiting commenced at once and was urgent. At first it was brown and fæcal-smelling, but later it became green. On the third day of the attack it diminished, and only occurred after food. She did not vomit after admission. The bowels were opened naturally the day before the attack, and on every subsequent day by enemata.

On admission the patient was in good condition. Her pulse was 90 per minute, and her temperature 98° F. She had an icteric tinge, but no definite jaundice. She was not vomiting. A turpentine enema was returned with a small result, but no flatus. The pain was definitely peristaltic in character, but no dilated coils of gut were seen or felt. The abdomen was not distended. There was no rigidity or tenderness even in the epigastric region or around the gall-bladder. No tumour was felt. On vaginal examination nothing abnormal was found. Douglas's pouch was empty. The rectum was ballooned.

A diagnosis of gall-stone intestinal obstruction was made, as the notes state, for the following reasons:

1. She is a fat woman, aged forty-nine, with acute intestinal obstruction without distension, and therefore of the small gut.

2. The gut is not strangulated, because the pain is peristaltic and intermittent, and this can only be explained by a gall-stone, an incarceration beneath a slack band, or a large pouch.

3. The vomiting was at first black, fæcal, and urgent. It became green and bilious on the second day, and diminished on the third day, when it ceased—*i.e.*, stage 1 and 2 of typical vomiting in gall-stone obstruction.

4. The bowels have been opened day by day in response to enemata, and a little fæcal matter has come away with each of two enemata given in hospital.

5. There is a history of chronic hypochondriac pain, and the patient is now a little jaundiced.

Operation.—The abdomen was opened below the umbilicus. Clear fluid was found in the peritoneal cavity. The small intestine which presented was congested and dilated. A hand passed down into the pelvis immediately encountered the stone, which was drawn out of the abdomen in the ileum and packed round with sponges. The situation of the stone was not certainly ascertained, but it was in the lower ileum. There was no well-marked ring of spasm below the stone, as in previous cases. A longitudinal incision was made on the stone, which was pressed out, and the hole immediately closed with two layers of No. 1 silk continuous suture. The inner was through all layers; the outer was a Halsted invaginating suture. The gut was washed with saline and returned. The abdomen was closed with through and through silkworm-gut sutures for the sake of speed.

After-Treatment.—The patient's condition was very good after the operation, but, taught by the last case, she was stimulated from the first with coffee-and-brandy enemata, and strychnine ($\frac{1}{60}$ grain) and caffeine (2 grains) hypodermically, every four hours.

She made an uninterrupted recovery, and healed by first intention. It was egg-shaped, and measured 2 inches in length and $1\frac{3}{16}$ inches in diameter.

Case VI.—Intestinal obstruction in a woman, aged seventy-three, for four days, due to a gall-stone 1$\frac{1}{16}$ inches in diameter, impacted in ileum 10 feet above the cæcum. Enterotomy; recovery.

S. S. was admitted to the London Hospital on October 6, 1904. She was an emaciated old female, and her history was far from definite. She had suffered from epigastic pain for years, but had never had jaundice or biliary colic. Four days before admission she had fairly severe epigastric pain, but she had taken no purgative. Three days before admission she was suddenly seized with very severe pain all over the abdomen. This pain, she stated, was continuous, and was not colicky in character. Vomiting followed immediately, and was persistent until she was admitted to the hospital, when it was fæcal, frequent, and gushing in character. She had suffered much from flatulence, but all the wind was ejected from the mouth, and she had passed none by rectum. She had been totally constipated since the onset of the pain, in spite of several purgatives and enemata administered by her doctor.

Present Condition.—Her general condition was fair. Her pulse somewhat above 100, but strong. Her temperature normal. She was one of those thin, aged women who bear operation so well. There was no jaundice. The abdominal examination revealed nothing, except, perhaps, a little resistance and tenderness in the gall-bladder region. No tumour could be felt in Douglas's pouch either by the rectum or vagina.

Diagnosis.—A probable diagnosis of gall-stone intestinal obstruction was made, but the history was so inconclusive on the subject of pain, vomiting, and constipation, that it was obviously not a case for dogmatism.

Operation. — The stomach was washed clean with a warm solution of bicarbonate of soda before the operation. The abdomen was opened in the middle line below the umbilicus. All the small intestine which presented was collapsed. The ileum was then swiftly followed up from the ileo-cæcal valve for a distance estimated at 10 feet, when a large gall-stone in the gut was encountered. The gut was typically dilated above and contracted below the stone. Gut clamps were applied and sponges packed round. The stone was cut on longitudinally and evacuated, and the gut closed in two layers, as in the previous cases. One or two Lembert sutures were applied in the middle of the incision, which appeared weak. The gut was washed in saline and reduced. The abdomen was closed with through and through silkworm-gut sutures. The whole operation occupied seventeen minutes.

After-Treatment.—The patient had 4 pints of saline transfused subcutaneously into the thighs, and was stimulated with strychnine ($\frac{1}{60}$ grain) and caffeine (2 grains) alternately every two hours.

On the day after the operation she was free from pain and in very good condition, and had vomited but once. Her bowels had, however, not been open, and this so excited the misdirected zeal of my house-surgeon that he prescribed 10 grains of calomel in doses of a grain an hour.

On the second day I saw her again. She had vomited four times in the night after the calomel, and five times since morning. The vomited matter was curdled milk, and was not bile-stained. She was jaundiced. There was no sign of peritonitis in the abdomen, her pulse was 84 beats per minute, and her temperature normal. I thought her condition due to gastritis and duodenitis, due to the calomel. All food was stopped by mouth except iced champagne. Nutrient enemata were ordered for twenty-four hours, and bismuth and sodium bicarbonate were prescribed. Later in the day her bowels were open four times and the vomiting ceased. On the following day feeding was recommenced with white of egg and beef-tea. She then made an uninterrupted recovery, and healed by first intention.

It will be gathered that I am strongly opposed to the post-operative exhibition of small doses of calomel frequently repeated. Had this old woman not possessed remarkable powers of endurance, she would certainly have succumbed to this maltreatment.

If the desire to see the bowels moved soon after an operation for intestinal obstruction is overpowering, a small ½ pint soap-and-water enema may be prescribed without risk.

The stone removed measured 1⅝ inches in length, and 1$\frac{1}{16}$ inches in its greatest diameter.

During the years 1893-1905 there have been eight further cases of intestinal obstruction due to gall-stones admitted to the London Hospital. For permission to publish the following summaries of these cases I am indebted to the kindness of my colleagues.

Case VII.—Intestinal obstruction in a woman, aged seventy-three, for four days, due to a gall-stone 1 inch in diameter, impacted in the ileum 1 foot above the cæcum. Enterotomy; death.

H. G., aged seventy-three, was admitted on January 3, 1894, with a diagnosis of acute intestinal obstruction, under the care of Mr. T. H. Openshaw. She was a very fat married woman. Four days before admission she was suddenly seized with paroxysmal pains in the region of the umbilicus, and these pains had persisted. Vomiting had been constant for three days, but on the fourth day of her illness it remitted. At first it had been black, then green, and finally brown and fæcal-smelling. Constipation had been complete for five days. The abdomen was a little distended. No other sign was made out.

Operation.—Laparotomy was performed, and a gall-stone found in the ileum 1 foot from the ileo-cæcal valve. The wall of the intestine was thin and nearly ulcerated through. An attempt was made to break up the calculus with a needle passed through the intestinal wall below, but this failed. The gall-stone was then removed by a longitudinal incision on it, and the wound closed by a continuous suture for the mucous membrane, and twelve fine catgut Lembert sutures for the peritoneum. The patient did not rally from the operation, and died four hours later. No post-mortem was performed.

The calculus was the shape of a walnut, 1½ inches in its longest diameter and 1 inch in its shortest. It was whitish in colour and semi-opaque, and consisted entirely of cholesterin with no incrustation. It was studded with whitish elevations.

CASE VIII.—Intestinal obstruction in a man, aged forty-two, for four days, due to a gall-stone 1 inch in diameter, impacted in the ileo-cæcal valve. Enterotomy; recovery. This gall-stone was felt before operation, and had probably remained in the gut ten years.

G. S., aged forty-two years, was admitted to the hospital, under the care of Mr. F. S. Eve, on March 19, 1894, suffering from intestinal obstruction. His previous history was of very great interest. In 1875, eighteen years before, he had a severe attack of pain in the epigastrium, accompanied by tenderness and vomiting. During the next seven or eight years he was never entirely free from pain—*i.e.*, until 1884. He had severe attacks of pain about once every week during this period. He then consulted an eminent physician, who treated him for dyspepsia. Six months later the pain almost entirely disappeared, but was replaced by an obstinate constipation, making it necessary for him to take purgatives once or twice a week. Unless the bowels were open daily, the patient had a pain low down in his abdomen.

Mr. F. S. Eve is of opinion that during the whole of this period of ten years (1884-1894) the gall-stone was in the small intestine. On March 15, 1894, four days before admission, severe pain, with continuous vomiting and absolute constipation, suddenly appeared. Under an anæsthetic, a hard, bullet-like lump was felt in the right iliac fossa. Laparotomy was performed on March 20 by Mr. F. S. Eve, as no result had followed the use of enemata. A gall-stone was found impacted at the ileo-cæcal valve. This was displaced up the ileum in order that the incision might be through uninjured gut. The stone was then cut on and removed. The mucous membrane was first sutured, then the wound was invaginated by a continuous Lembert suture, and finally a piece of omentum was drawn over the incision and fixed by sutures. The gall-stone was 1½ inches long, 1 inch in diameter, and 3¼ inches in circumference, and weighed 190 grains. It had no facet. It was of a dirty white colour, and its surface, which is washed and worn, tuberculated and friable, is largely formed of phosphates. Indeed, it presents all over the appearance of the ends of the two calculi in my case which had projected into the intestine from the gall-bladder. This calculus is now in the museum of the London Hospital.

Progress.—On the eleventh day after the operation, some thick, foul, yellowish-green pus discharged through the wound from a stitch abscess. The wound soon healed, however, and the patient left the hospital, cured, seven weeks after the operation.

This case was published in the Clinical Society's Transactions for 1895, vol. xxviii., p. 91, with an analysis of twenty other cases.

Case IX.—Intestinal obstruction in a woman, aged sixty-three, for four days, due to a gall-stone 1 inch in diameter, impacted in the ileum several feet above the cæcum. Enterotomy; recovery.

E. S., aged sixty-three years, was admitted to the hospital under the care of Mr. Jonathan Hutchinson, junior, on November 6, 1895, with acute intestinal obstruction.

History.—She gave a clear history of attacks of jaundice and biliary colic. On November 2, 1895 (four days before admission), she had been seized with sudden and acute pain in the right hypochondriac region just below the ribs, and with vomiting. The bowels were opened on the following day, but for the next two days only flatus was passed, and the vomiting was continuous. On the day before admission the vomiting became fæcal and the constipation absolute. On admission the patient looked very ill, and she was slightly jaundiced. Her pulse was rapid and small. The abdomen was tender and distended. Laparotomy was performed on the day of admission, and a gall-stone was found several feet above the ileo-cæcal valve. The gut above was greatly distended and congested; below it was contracted and empty. Enterotomy was performed, and the stone evacuated through the wound; two small stones were found above it, and these fitted facets on the larger one. The *stone* which caused the obstruction was 1¾ inches long, 1 inch in diameter, and 3⅛ inches in its greatest circumference. It weighed 191 grains. It was dark brown, and at its larger end presented four facets. The two smaller stones presented four facets each. It was clear that two or more other calculi were missing. The wound in the gut was closed in a similar manner to that described in the previous cases.

Progress.—The woman made an uninterrupted recovery, saving a sudden attack of collapse on the fourth day after the operation, for which no cause was found, and from which she rallied. She was discharged quite well from the hospital a month later.

This case was recorded in the Pathological Society's Transactions, 1896, vol. xlvii., p. 95.

Case X.—Intestinal obstruction in a man aged fifty-two, for fourteen days, due to a large gall-stone impacted 25 inches above the cæcum. No operation; death. Diagnosis complicated by incarcerated inguinal hernia.

H. S., a man aged fifty-two years, was admitted to the London Hospital on October 24, 1898, under the care of Mr. McCarthy.

He was in a state of profound collapse, and presented a large incarcerated right inguinal hernia, in which there was scarcely any impulse on coughing. For fourteen days he had been vomiting, and latterly this had been fæcal. For two days he had suffered from absolute constipation. He was too collapsed for an operation to be performed. He died the day after admission.

Post-mortem.—A large gall-stone was found impacted in the ileum 25 inches from the cæcum. A fistula bimucosa connected the gall-bladder and duodenum just beyond the pylorus, and these structures were united by peritoneal adhesions. Commencing peritonitis was present all over the small intestine. He had a fatty heart and a horseshoe kidney with two ureters.

CASE XI.—Intestinal obstruction in a woman, aged fifty, for ten days, due to a gall-stone in the ileum. Enterotomy; death. Possibly this stone had been in the gut twelve months.

S. N., aged fifty years, was admitted, under the care of Mr. T. H. Openshaw, on November 27, 1899, suffering from acute intestinal obstruction.

Previous History.—She had never had an attack of jaundice. Twelve months before admission she had a severe attack of abdominal pain, which came on suddenly and was spasmodic in character. This was accompanied by vomiting. During the year she had six or seven similar attacks of spasmodic abdominal pain, accompanied by vomiting, and each of about two days' duration. The present attack commenced ten days before admission with "spasms" and sickness. The constipation was absolute throughout; neither fæces nor flatus passed. On admission the vomiting was fæcal, and had been so for six days. The abdomen was greatly distended.

Operation.—A gall-stone was removed from the ileum by an incision directly over the stone. The wound was closed by a double layer of sutures, the outer being Lembert's. The patient died forty-eight hours later.

Post-Mortem.—The wound in the gut was found to be soundly united and watertight. A fistula bimucosa the size of a shilling connected the gall-bladder with the first part of the duodenum, these parts being firmly united by peritoneal adhesions. Two other gall-stones were found in the gall-bladder, one the size of a filbert-nut, the other of a pea.

CASE XII.—Intestinal obstruction in a woman, aged sixty, due to a gall-stone 1⅛ inches in diameter, impacted in the ileum 3 feet above the cæcum. Enterotomy; death.

Catharine Hicks, a very fat married woman aged sixty, was admitted into the London Hospital, under the care of Mr. Jonathan Hutchinson, junior, on May 9, 1900. No notes were taken of this case before operation.

Operation.—A low median laparotomy was performed, and a large truncated gall-stone was found impacted in the ileum, 3 feet from the cæcum. The gut was firmly contracted below it and dilated above. The calculus was pushed up 1 inch and removed, as in the previous cases, and the incision closed. Two smaller gall-stones were found above the larger one, and another small one was discovered after the bowel had been closed. This was pushed past the ileo-cæcal valve. The patient did not rally, and died in a few hours.

Post-Mortem.—A round hole was found in the first part of the duodenum, leading into the gall-bladder, and surrounded by dense adhesions. The small intestine showed commencing peritonitis. The heart was fatty, the aorta atheromatous, and the kidneys granular.

The *calculus* was in shape a truncated cone, 1¼ inches long, and 1⅛ inches at its base, and ⅝ inch at its apex, in diameter.

CASE XIII.—Intestinal obstruction in a woman, aged sixty, for five days, due to a gall-stone the size of a hen's egg, impacted midway between duodenum and cæcum. Enterotomy; death.

Sarah Quittenton, a very fat woman aged sixty, was admitted to the London Hospital on August 17, 1901, under the care of Mr. T. H. Openshaw.

The onset of her illness was not sudden. She had been ill for some weeks, but able to get about until five days before admission. She had been constipated for fourteen days, and vomiting for six days. The vomit was fæcal on admission. The abdomen was not distended.

Operation.—A gall-stone the size of a hen's egg was removed from the small gut, and the opening and abdomen closed in the usual way. The patient died twenty hours later.

Post-Mortem.—The gall-bladder was adherent to the first part of the duodenum and to the transverse colon. It was small and contracted, and communicated with the duodenum by a fistula bimucosa which admitted the index-finger. The enterotomy wound in the gut was midway between the duodenum and the cæcum. The heart was fatty and flabby, and the aorta atheromatous.

CASE XIV.—The spontaneous passage of three large faceted gall-stones after nineteen days' obstruction. Death nine days later from fatty heart, granular kidneys, and emphysema. For the notes of this case I am much indebted to my colleague, Mr. T. H. Openshaw.

Amelia Collingborn, an extremely fat married woman aged sixty-three years, was admitted to the London Hospital on December 21, 1901. The history was very incomplete as the patient was delirious and suffering from fæcal vomiting. She had not been well for five weeks, and her bowels had not been open for a fortnight. She had suffered from diffuse pain over the abdomen, and the vomit had been fæcal for several days. The treatment was expectant. Her bowels were open by enemata during the first two days after admission. She then suffered from incontinence of fluid fæces.

On December 26, 1901, and five days after admission, she passed two large faceted gall-stones $\frac{3}{4}$ inch to $\frac{7}{8}$ inch in diameter, and on the following night she passed another. On the sixth day she could answer questions, but after this her temperature became subnormal and she gradually sank, and died on the tenth day after passing the stones.

The following notes are taken from Dr. Schorstein's account of the autopsy:

The subcutaneous fat on the abdomen was $2\frac{1}{2}$ inches thick. The pericardium, both visceral and parietal, was covered with fat. The heart weight 13 ounces, the cardiac muscle was soft and friable, and fatty infiltration was present. The endocardium was stained with blood-pigments. The lungs were emphysematous. The cortex of the kidneys was greatly reduced. A fistula was found between the gall-bladder and the duodenum, 1 inch from the pylorus and $\frac{3}{4}$ inch in diameter, leading into an empty gall-bladder. The three calculi varied in diameter from $\frac{3}{4}$ inch to $\frac{7}{8}$ inch. One was cylindrical with flat facets transversely placed at either end. One was cylindrical with flat facets obliquely placed at either end, so that it was triangular when viewed from the side. The third was roughly pyramidal in shape. The facets indicated the presence of two or more gall-stones in the gall-bladder.

This case illustrates the possibility of the spontaneous passage of very large gall-stones through the ileo-cæcal valve, and further elucidates the very high mortality in this class of intestinal obstruction; for these cases are, with very few exceptions, some of the worst subjects for operation a surgeon is called to treat.

2. INTERNAL STRICTURE OF THE INTESTINE.

Definition.—An internal stricture is a narrowing of the lumen of the bowel produced by changes in the wall of the intestine.

The lumen may be diminished in three ways: (1) It may be blocked by new growth projecting from the wall, as is seen in cancerous stricture. (2) The wall itself may be contracted, and this is usually the result of healing ulcers and scar formation from gangrene and trauma. (3) Finally, the wall may be so greatly thickened by the deposit of new tissue in it that the lumen of the bowel is greatly narrowed. This new tissue is either cancerous, tubercular, or gummatous.

It is customary to exclude strictures of the rectum from a description of intestinal obstruction.

Internal strictures of the intestine are divided into (i.) carcinomatous, (ii.) cicatricial, and (iii.) congenital.

(i.) **Carcinomatous Stricture of the Intestine**—*Incidence.*—Cancer of the intestine is second only to intussusception as a cause of intestinal obstruction. Of the 669 consecutive cases of obstruction at the London Hospital (1893-1905), 151 were due to cancer.

Etiology.—The primary cause of cancer is, no doubt, the same in the intestine as elsewhere, and it is equally unknown. The predisposing causes are apparently trauma, chronic inflammation and irritation, stagnation of intestinal contents, and senility of the tissues. The influence of trauma, inflammation, and irritation, are well marked in the distribution of cancer of the intestines. In the sigmoid flexure and the rectum, where the contents are solid, it is much more frequent. Stagnation appears to play a part by increasing the time during which the contents can act by trauma and irritation upon the intestinal wall. The contents loiter in the duodenum, cæcum, and the sigmoid flexure and rectum, and these are all special sites for cancer. Nothnagel believes that cancer not infrequently commences in the base or scar of an ulcer, and Dr. H. D. Rolleston has collected ten cases recorded by Letulle, Perry and Shaw, and Nathan Larrier.

The influence of age is not so well marked in cancer of the intestine as in cancer elsewhere. The majority of cases occur between 40 and 65 years of age, but Nothnagel has collected no fewer than 61 cases between 20 and 30 years, and mentions other instances at 17, 13, 12, 11, 3½, and even 3, years of age. Maydl states that one-sixth of all cancers of the intestine occur between 30 and 40 years, and one-seventh before 30 years. Cancer must not be excluded, therefore, when the diagnosis of an intestinal stenosis is being considered in the case of a young adult. A specimen of colloid carcinoma of the sigmoid flexure in a boy aged ten years was recently sent to the Pathological Department of the London Hospital by Dr. Milne, and cases of colloid cancer of the colon at an early age have also been put on record by Kanthack and Furnival, and by W. A. Garrard. Sex appears to have little influence on the occurrence of cancer of the bowel. Of the 151 cases which caused obstruction at the London Hospital, 65 were men and 86 were women.

The Pathology of Cancer of the Intestine.—Carcinoma of the intestine is nearly always primary. Secondary deposits of cancer from growths elsewhere are most unusual, but this remark does not apply to melanotic growths in the same degree (Thomson). Cancer of the intestine by extension from the stomach, gall-bladder, pancreas, and female genital organs, is not so rare, but is treated of under Compression of the Intestines.

Histology.—Primary carcinoma of the intestine always originates in the glands of Lieberkühn, and is called columnar-celled carcinoma, malignant adenoma, cylindrical carcinoma, or cylindroma. When the epithelial elements grow rapidly and preponderate, the tumour is large, soft, and brain-like in consistency, and is known as "medullary" or "encephaloid" carcinoma. In other cases the tumour undergoes colloid degeneration, and the growth is then called "colloid carcinoma." Very rarely there is a well-marked cicatricial reaction of the connective-tissue matrix in a slowly growing tumour, and a scirrhous carcinoma is then produced.

The Situation of the Carcinoma.—Primary carcinoma of the intestine is exceedingly rare in the small gut. The great majority of the cases are located in the sigmoid flexure and the rectum. The cæcum and ileo-cæcal valve provide a small but well-marked group. The duodenum, in spite of its brevity, is responsible for about half the cancers of the small intestine. To illustrate these points I have analyzed the distribution of all the cancers of the alimentary canal which are contained in the medical and surgical records of the London Hospital from 1900 to 1905. They amount to 1,004 cases. Nothnagel's figures are appended for comparison :

Source.	Œsophagus.	Stomach.	Duodenum.	Jejunum.	Ileum.	Small Gut.	Appendix.	Cæcal and Ileo-Cæcal.	Ascending Colon.	Hepatic Flexure.	Transverse Colon.	Splenic Flexure.	Descending Colon.	Sigmoid Flexure.	Rectum.
London Hospital Medical and Surgical Registers ...	145	378	5	3	2	5	—	41	6	3	17	12	6	103	278
Nothnagel Pathological Institute, Vienna:															
1870–1881	—	—	2	—	4	—	1	* 9	6	—	17	—	—	13	48
1882–1893	—	—	5	—	6	—	1	14	—	—	63	—	—	40	114

* Colon in general.

Secondary deposits from carcinoma of the intestine occur later less frequently than in cancer elsewhere. The prognosis after early diagnosis and excision is therefore relatively good. It is said that this statement does not apply to the rare cases of cancer of the small intestine. Metastasis is most common in the lymphatic glands, but it is nearly as frequent in the liver. After these the peritoneum, omentum, and mesentery, are most often invaded, and then the lungs and kidneys. Hauser states that the ordinary and scirrhous form of cancer of the intestine chiefly produces deposits in the liver. The colloid form, on the other hand, has a great tendency to invade the peritoneum, whilst the metastases of encephaloid cancer are mainly in the lymphatic glands.

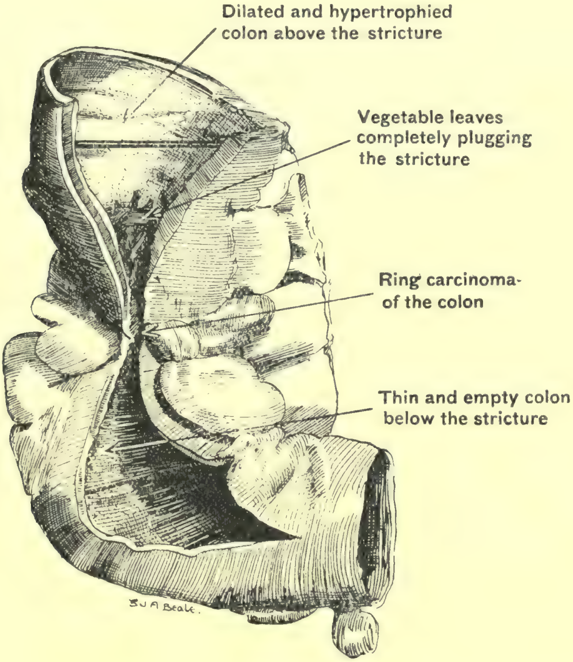
Dilated and hypertrophied colon above the stricture
Vegetable leaves completely plugging the stricture
Ring carcinoma of the colon
Thin and empty colon below the stricture
S. J. A. Beale.

FIG. 10.—FROM A SPECIMEN IN THE LONDON HOSPITAL, SHOWING A TIGHT RING CARCINOMA OF THE SIGMOID FLEXURE PLUGGED WITH VEGETABLE LEAVES.

The Anatomical Forms of Cancer of the Intestine.—The form in which a cancer is found at an operation or an autopsy depends upon the time that it has existed and the rate at which it has grown. Those forms which produce obstruction as a rule come under observation early, whilst those that do not interfere with the lumen of the bowel are only seen at an advanced stage.

It is, however, of practical importance to recognize several different varieties, because the effects and symptoms produced by them are very different, and the physician must be prepared to recognize cancer of the intestine in all its protean forms.

1. *The Nodular Variety.*—It is probable that all cancers commence as a small nodule in the deeper part of the intestinal mucous membrane, but at this stage they can only be discovered by accident.

2. *The Polypoid Variety* develops from the last. The nodule grows towards the lumen of the bowel, and very exceptionally may become pedunculated. The mucous membrane for a time is intact over the growth, but later it is ulcerated and the tumour fungates. Such a malignant polypus may produce an intussusception, but this is rare as compared with innocent polypi.

3. *The Annular or Ring Carcinoma.*—This is one of the commonest forms in which cancer of the intestine is found (*vide* Fig. 10). The bowel is often puckered in as though a string had been tightly tied around it, and the actual mass of the cancer is very small. As a rule only one stricture exists, but several secondary ones may be present. The annular arrangement, which is well marked in all cancers of the bowel, is due to the cancerous infection following the lymphatics which run round the intestine to the mesentery. Some carcinomata, however, appear to have started in the scar of a stercoral ulcer, especially in the sigmoid flexure. These scars often surround the intestine, and at first the cancerous process is limited to the scar. The diagnosis is confused in such a case by the long history of the chronic constipation, followed by simple ulceration and stricture which preceded and produced the cancer. Even at the operation or autopsy, the microscope alone can determine in many cases whether the stricture is malignant or not. The lumen of a malignant stricture is, however, usually ulcerated.

The symptoms of ring carcinoma are those of progressive stenosis of the bowel, unless a foreign body suddenly becomes impacted, when the onset of symptoms may be very acute and sudden.

4. *The Rigid Tube.*—The cancerous process has in some cases involved some 2 or 3 inches of the intestine, and converted it

into a rigid tube (*vide* Fig. 11). The lumen is as a rule ulcerated and patent. The signs of intestinal obstruction are not well marked in such cases, and the symptoms are obscure. Under

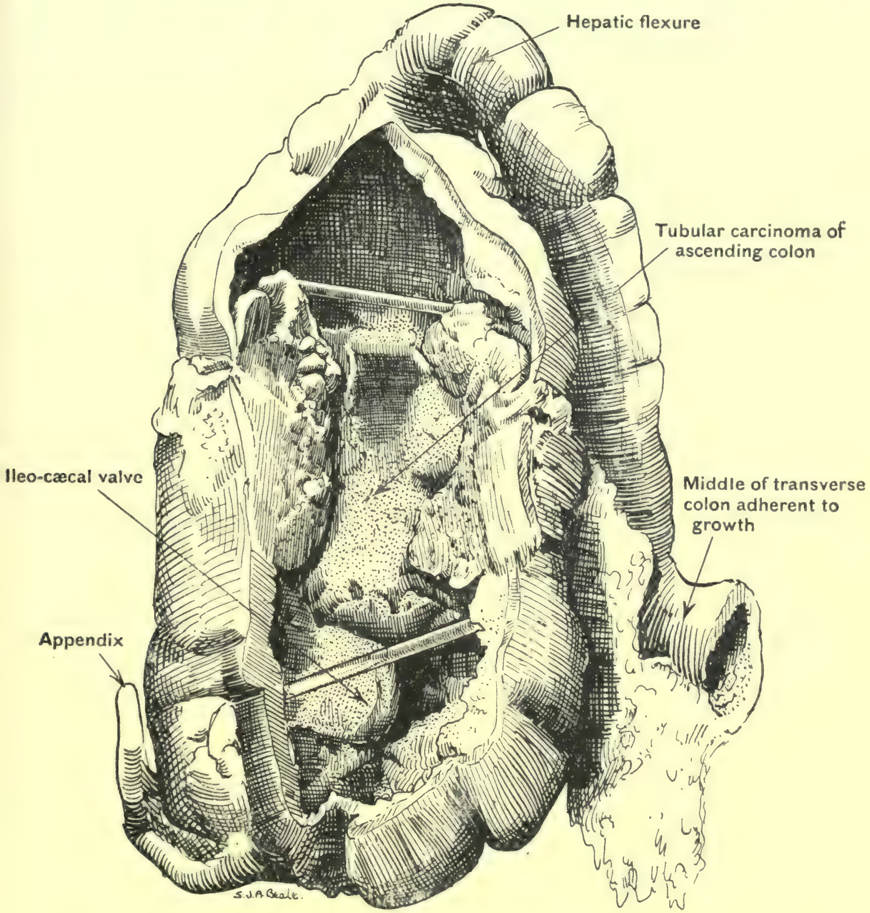
Hepatic flexure
Tubular carcinoma of ascending colon
Ileo-cæcal valve
Middle of transverse colon adherent to growth
Appendix
S.J.A.Beale.

FIG. 11.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM, SHOWING A TUBULAR ULCERATING CARCINOMA OF THE ASCENDING COLON REMOVED BY THE AUTHOR.

The patient was alive and well two and a half years later.

Intussusception it is pointed out that a cancer which has produced invagination often spreads in the middle layer, and converts it into an inverted cancerous tube.

5. *Papillomatous or Cauliflower Variety.*—This also is a common form of cancer of the intestine. When the growth of a cancer is rapid, it passes quickly through the nodular and perhaps the polypoid forms, and projects into the bowel as an exuberant cauliflower-like mass of friable growth. At first it is attached only to a part of the circumference of the gut, and this stage is a very favourable one for removal. The sigmoidoscope enables such tumours to be clearly seen, and their mobility ascertained as high up as the middle of the sigmoid flexure (Mummery).

The actual obstruction caused by such growths is slight, and is limited to some abdominal unrest and flatulence, but the wind passes freely and without much delay. Tenesmus is often present, and, as the growth is nearly always ulcerated, some blood and mucus are frequently passed. When the growth is situated in the lower part of the colon, the patient frequently has the desire to void the discharge, which has accumulated during the night, directly he rises in the morning, a symptom known as "morning diarrhœa," which is very characteristic.

6. *A Large Tumour.*—When a case of cancer of the intestine does not terminate early as a result of intestinal obstruction, acute peritonitis, or extensive metastases, the primary growth develops locally into a large mass of new growth. The growth surrounds and infiltrates the wall of the intestine, and invades the adjacent cellular tissue and neighbouring organs. It fills the lumen of the gut with a fungating, exuberant, and ulcerating mass from which a profuse discharge is poured out. In rare cases the primary growth is of moderate size, but the major part of the tumour is formed of a vast mass of secondary lymph-glands in the mesocolon. Such large masses of new growth are closely simulated by accumulations of solid fæces above the stricture, or by coils of gut matted around the growth by local peritonitis, in which localized abscesses may occur. Attempts should be made by means of enemata and laxatives to remove such fæcal accumulations, but this is no easy matter.

7. *The Ulcerating and Gangrenous Form.*—All forms of cancer of the intestine ulcerate, but in some the process is so active as to merit the above description. The low vitality of the tissue, which is constantly bathed in corrosive fluids swarming with micro-organisms, accounts for the ulceration. This condition is indicated by a profuse and frequent diarrhœa and tenesmus. The stools contain blood and pus in large quantities, and when the process becomes gangrenous they are inexpressibly foul, and

frequently contain rotten masses of new growth. Cancerous cachexia is usually well marked in such cases, and is due to the loss of fluids from the raw surfaces and the absorption of the products of decomposition. It is, however, remarkable how little cachexia may be present in some very extensive cases of ulceration, and this may be attributed to the absence of lymphatics in new growths.

The onset of ulceration in an annular growth may open the passage so that the symptoms of chronic stenosis which previously existed are suddenly relieved and replaced by a profuse diarrhœa, and the condition of the patient may in many ways improve.

The Secondary Changes and Results produced by Carcinoma of the Intestine.—These are as follows :

1. Chronic stenosis of the intestine.
2. Acute dilatation of the colon above the stricture.
3. Acute dilatation of the cæcum.
4. Intussusception.
5. Vovulus, bending, kinking.
6. Peritonitis — Localized : (*a*) adhesive, (*b*) suppurative.
   General : (*a*) perforative, (*b*) due to transudation.
7. Fistulæ.
8. Ballooning of the rectum.

1. *Chronic Stenosis of the Intestine.*—Cancer of the intestine is the most frequent and typical cause of chronic stenosis; and as the pathological changes and symptoms of this condition have been fully described earlier, it will not be necessary to repeat them here. The pathological changes are chiefly in the intestines above the growth, and they may be briefly stated to be dilatation, hypertrophy, lengthening of the intestine, inflammation and ulceration, and tension striæ in the mesentery.

2. *Acute Dilatation of the Colon.*—This is a very common termination to a case of cancerous stricture. Within a few hours a case which was progressing apparently favourably may become hugely distended, profuse vomiting may appear with absolute constipation, and death may follow in a day or two. At an operation or autopsy the colon is found to be so enormously distended that the longitudinal striæ have disappeared, and this distension may extend to the ileum, and even to the jejunum, duodenum, and stomach. The tension of the intestinal wall is so great that they readily rupture, especially at the points where patches of gangrene

and softening appear. The colon, as it dilates between the layers of its mesentery, shortens that structure and anchors the gut. The contents of the colon are usually fluid in the part adjacent to the stricture, although the upper parts often contain much gas.

The causes of this sudden distension are various, but may be described as a failure of the compensation offered by a progressive hypertrophy to an increasing stenosis of the intestine. The lumen of the stricture has in some cases been found completely occluded by a foreign body, such as a fruit-stone, or, as in a case of my own, by tea-leaves, so that it offers an insuperable obstacle to the most powerful waves of peristalsis. In many cases acute dilatation has been precipitated by the unwise administration of purgatives. The colon, which was already loaded to the limit of its powers, has suddenly had to receive from above some pints of fluid fæces, and compensation has broken down altogether. In other cases the muscular wall of the intestine adjacent to the stricture has apparently become exhausted and paralyzed, and has been inflated by the active intestine above it. In some instances general peritonitis is found originating in the patches of gangrene and this no doubt assisted in producing the sudden distension.

3. *Acute Dilatation of the Cæcum.*—In this condition the cæcum suddenly becomes enormously dilated, whilst the rest of the colon above the cancerous stricture is usually only moderately distended, and is not tense. Gangrenous patches appear in the cæcum within a few hours, and are usually referred to as stercoral ulcers. The general peritoneal cavity is infected from these patches. In the worst cases the cæcum bursts and collapses, whilst the peritoneum becomes distended with free gas and fluid fæces (pneumoperitoneum). More usually the cæcum becomes adherent to neighbouring structures by recent lymph, which prevents actual extravasation until these adhesions are separated. The carcinoma is usually of the ring or annular variety, and acute dilatation of the cæcum is often the first sign of the presence of the cancer. Of my six cases, three had been in perfectly good health until ten days, six days, and two days, before admission to the hospital. The distribution of the stricture in my six cases was in the sigmoid in three cases, the splenic flexure in two, and one in the hepatic flexure. It will be seen that an undue proportion of cases is in the upper colon. The mechanism of the acute dilatation of the cæcum is obscure. It cannot be due to paralysis, as is usually

stated, for this will not account for the tension on the walls, the pressure-gangrene, and even the rupture of the organ.

It is clear the cæcum is dilated or distended by the colon below

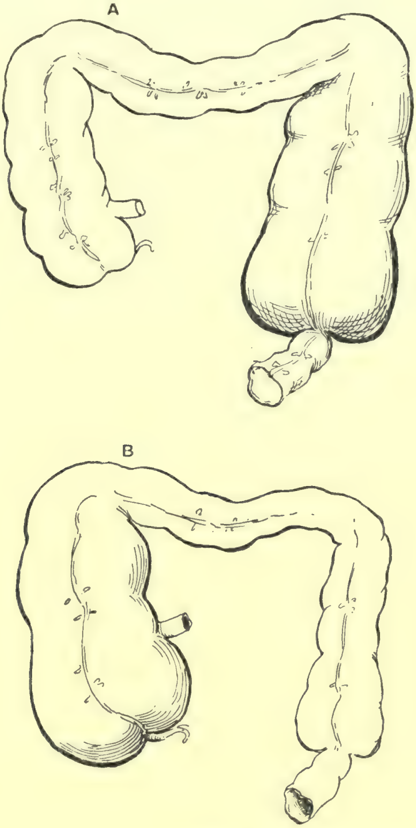

Fig. 12.—To illustrate the Method of Production of Acute Dilatation of the Cæcum.

In A is shown the usual condition found when acute obstruction supervenes in a stricture of the sigmoid flexure. The colon above the stricture is dilated and hypertrophied for a greater or less distance. In B is shown the condition found in acute dilatation of the cæcum. The cæcum and ascending colon are enormously dilated. The rest of the colon down to the stricture is about the normal size and is not tense.

it, which suddenly empties back its accumulated contents and blows up the cæcum like a child's balloon.

This emptying back may occur in three different ways. As the contents gradually accumulate above the stricture, the time comes when they are continuous as high as the cæcum. The colon just above the stricture is greatly hypertrophied, whilst the cæcum has not yet become so. When fluid continuity has been established, should the colon pass into spasm, the cæcum will be blown up by the more powerful muscle below it. This will not, however, account for the absolutely acute cases. In these we must either invoke antiperistalsis, which is a normal function of the upper part of the colon, or we may suppose that, whilst a forward wave of peristalsis is straining to force the accumulated contents through the stricture, the ring contraction which closes the colon behind the wave is burst open, and the contents are suddenly squirted back into the cæcum and distend it. The mechanism of the last explanation is similar to that of a gun which blows out its breech-block instead of propelling forward its projectile, and the devastation worked behind the gun finds its equivalent in acute dilatation of the cæcum (*vide* Fig. 12).

4. *Intussusception as a Result of Cancerous Stricture of the Colon* (*vide* Fig. 13).—Of 669 cases of obstruction at the London Hospital, 151 were due to cancer of the intestine, and of these only 5 produced intussusception. They were distributed as follows: Ileum, 1; ileo-cæcal valve, 1; sigmoid flexure, 2; upper rectum, 1. In two cases the growth was inverted, and formed the middle or returning layer. The form of the cancer may be lateral and polypoid, annular, or even tubular. In some cases the invagination leads to acute intestinal obstruction, but in the majority of cases the obstruction is partial and the symptoms subacute, or even chronic.

Reference may also be made to the section on Intussusception.

5. *Volvulus, Bending, and Kinking, as a Result of Carcinoma of the Intestine.*—Volvulus is a very rare occurrence, and no case has been recorded at the London Hospital. Bending and kinking are said to result from the distension above the stricture, combined with adhesions due to peritonitis around it, but these conditions are difficult to identify.

All these causes may produce an acute termination of a case of cancer which has been pursuing a chronic course.

6. *Peritonitis and Suppuration in the Cellular Tissue as a Result of Cancer of the Intestine.*—Infection is produced around a cancer either by a crude perforation or transudation of micro-organisms. Infection of the retro-peritoneal cellular tissues is rare, but it may

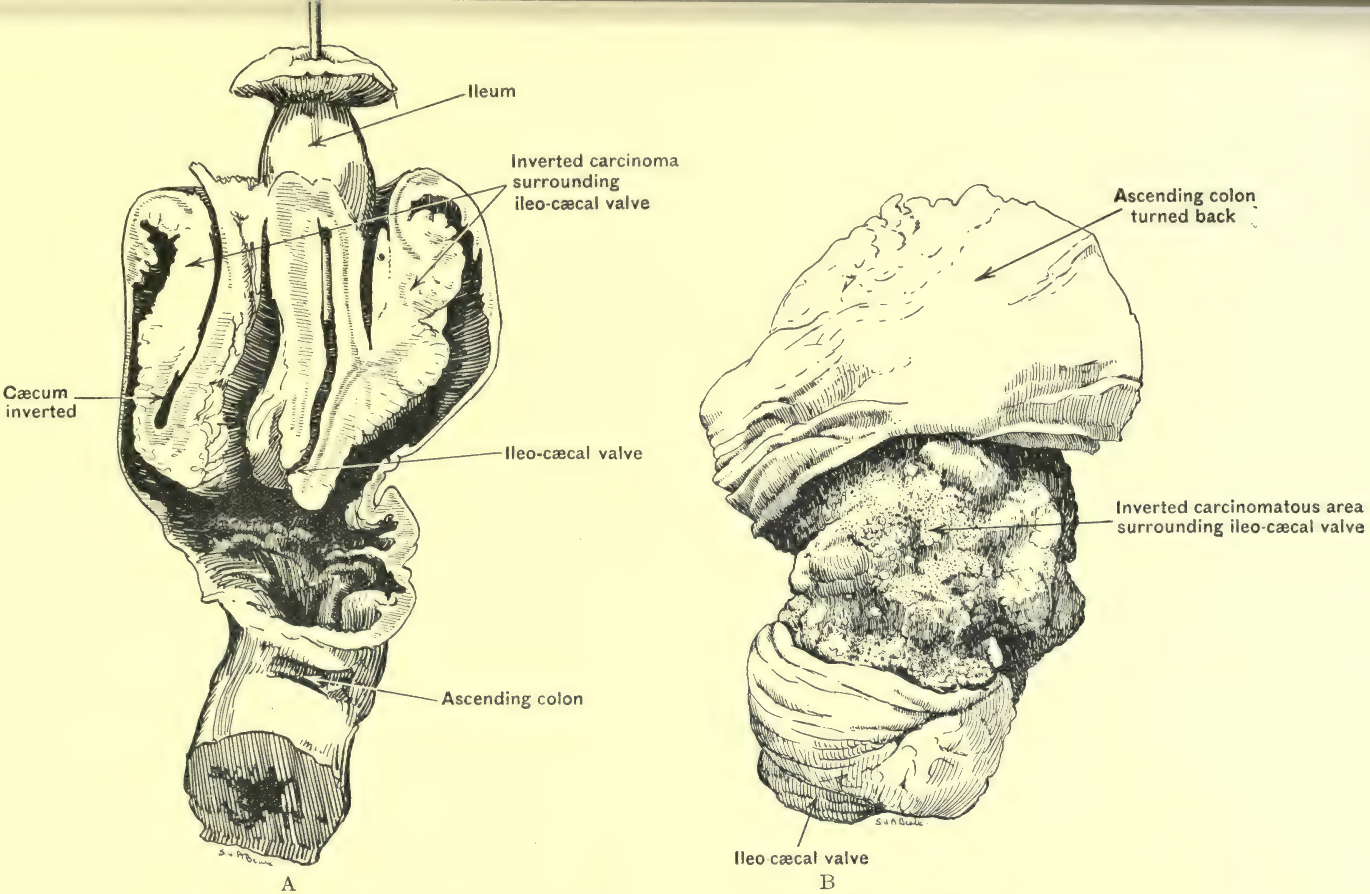
Ileum
Inverted carcinoma surrounding ileo-cæcal valve
Cæcum inverted
Ileo-cæcal valve
Ascending colon
A
Ascending colon turned back
Inverted carcinomatous area surrounding ileo-cæcal valve
Ileo·cæcal valve
B

FIG. 13.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM.
A circular area of carcinoma surrounds the ileo-cæcal orifice and has become invaginated into the ascending colon.

occur in any part of the colon. I have seen a stinking psoas abscess point in the left groin in a case where a cancer of the sigmoid flexure was adherent to the external iliac vessels, and had produced thrombosis of the iliac vein. In another case a large, hard mass in the left loin was found to be a suppurating mass of cancer connected with the descending colon.

Infection of the peritoneum is much more common. It may occur suddenly, as when actual perforation occurs and acute general peritonitis is produced, or gradually, in which case the peritonitis is more or less localized by adhesions. These adhesions may occupy only a segment of the abdomen, or may spread to all parts of it, producing general sclerosing peritonitis. Sometimes a single band, kink, or adhesion, is formed, which produces obstruction, and such cases may be operated on with success. A case of this sort was in the London Hospital, under the care of Mr. Mansell Moullin, in February, 1900. She had suffered from chronic stenosis for twelve months, and from acute intestinal obstruction for six days. Mr. Moullin found a coil of ileum adherent to a cancer of the cæcum. The peritoneum was studded with nodules. He divided the adhesion, and her acute symptoms were entirely relieved.

Suppuration may occur among localized adhesions, and chronic peritoneal abscesses may be produced. In the region of the cæcum, especially in the aged, it is difficult to distinguish such abscesses from appendicitis. In the lower sigmoid and upper rectum in women they produce a hard, indurated mass, easily mistaken for cancer of the ovaries or pyosalpinx unless the growth can be reached from the anus.

Acute general peritonitis is a common termination of cancer of the colon. As a rule a crude perforation is present. This may be in the growth, in a stercoral ulcer just above it, or in the cæcum. The late Dr. Slade pointed out that a suppurating gland in the meso-colon might also produce general peritonitis. The occurrence of general peritonitis in acute dilatation of the colon and cæcum has already been alluded to.

7. *Fistulæ as a Result of Cancer of the Intestine.*—When a cancer of the intestine becomes adherent to another hollow viscus, ulceration and perforation may follow, and a communication may be established. A fistula has thus been formed between the intestine and another part of the gut, the urinary bladder, the stomach, the vagina, or in rare instances the skin. A fistula between the intestine above and below the cancer has been known to relieve

the obstruction (Pollosson and Rolleston). The transverse colon may communicate with the stomach, so that fæcal matter is vomited and food is passed unchanged by the rectum. Almost any part of the colon may communicate with any other. The bladder most commonly communicates with the sigmoid flexure, but it may also be connected to the transverse colon or the cæcum. The operation of short-circuiting gives good results in the case of fistulæ, which will often close after the fæcal stream has been diverted.

8. *Ballooning of the Rectum as a Result of Carcinoma of the Intestine.*—This phenomenon is very often present in cancer of the lower part of the colon. It is, however, frequently found in other conditions, such as fæcal accumulations, appendicitis, pelvic tumours, peritonitis, and conditions of neurosis. I recently encountered a well-marked instance in a case of hæmorrhagic colitis. The features of the condition are as follows: When the index-finger is passed up the anus, it appears to pass through a hole in a septum into a large cavity filled with gas. The anal canal is shorter than is natural, and the tonic contraction of the sphincter tighter. When the finger is bent over to explore the interior of the cavity, this is found to wellnigh fill the pelvis, displacing the other organs, and to be about the size of a large orange. The mucous membrane is smooth and tense, and not velvety and rugose, as it should be. On reaching up to the roof of the cavity, in rare cases a cancerous growth may be felt. More frequently the upper part of the rectum is tightly constricted like a sphincter, and may even project into the cavity like an os uteri. The cavity is filled with gas, and on the several occasions on which I have introduced a rectal tube, this gas was expelled with considerable force, and the rectum collapsed. It is stated that ballooning of the rectum disappears under anæsthesia.

The explanation of this curious phenomenon has been much disputed. Sir Frederick Treves regards it as "due to some phase of paralysis," by which the bowel is dilated and fixed "like a pupil which has been dilated by atropine"; but it is difficult to conceive of a segment of the bowel so paralyzed that its walls are converted into a rigid shell capable of supporting the intra-abdominal pressure. The passage of a rectal tube which leads to the escape of the gas under pressure negatives this view, and we must conclude that the rectum is distended by flatus. It would appear that the irritation of the adjacent cancer produces spasm of the sphincter and corresponding relaxation of the detrusor of the rectum. Flatus therefore accumulates and distends the relaxed part of the rectum, which cannot expel it through the tight sphincter. It is also probable that the stricture acts as a strainer, retaining the solid and fluid fæces, but permitting flatus to pass freely.

The Symptoms of Cancer of the Intestine.—The clinical pictures which are presented by cancer of the intestine are of the most varied and different kinds, so that even in a description of its symptoms a pathological plan will be followed, and those will first be mentioned which are due to the cancer itself, whilst afterwards the signs and symptoms due to chronic stenosis and acute obstruction will be dealt with.

(*a*) *The Symptoms referable to the Cancer itself.*—A cancer of the intestine may remain practically latent for two or three years. The writer knew of a case where a medical man palpated and diagnosed a cancer of the rectum, but all operative treatment was refused. Nearly three years of an active and busy life elapsed before urgent symptoms of obstruction compelled a colotomy, and a huge cancerous mass was then found filling the pelvis. Latency is more likely when the growth is small, does not ulcerate or fungate, occupies but a part of the lumen, produces few metastases, and is situated low down in the alimentary canal. Cancerous cachexia is greater the nearer the growth is to the stomach.

The general symptoms of the presence of a malignant tumour are vague. They are a loss of strength, a sense of depression and abdominal discomfort, a progressive pallor and change in appearance of the face.

Pain is a prominent symptom in some cases, but it is usually due to peritonitis or obstruction. It is localized correctly when the tumour is in a fixed part of the bowel, but in the small intestine, transverse colon, or sigmoid flexure, it is as a rule referred to the umbilicus. In a few cases it has been "crossed," so that the pain of a cancer of the cæcum has been persistently referred to the left iliac fossa. In very rare cases the pain has depended on invasion of nerve trunks, such as the crural or sciatic nerves, and it is then neuralgic in character and widely diffused. In degree the pain of cancer of the intestine is not severe, apart from peritonitis and obstruction. It varies from a sense of discomfort to a continuous gnawing pain or the most severe neuralgia. When the pain bears some relation to the taking of food, the growth is often in the lower ileum or cæcum. When it is more severe before defecation, the growth is usually in the upper colon.

Tumour.—In about 40 per cent. of cases of cancer of the intestine, a tumour is palpable in the abdomen. Many parts of the bowel are inaccessible to palpation, whilst in other cases the tumour is small and covered by dilated intestine above it.

When a tumour is felt, it may vary in size from that of a nut to a child's head. In shape cancerous tumours are irregular, cylindrical, smooth, or nodular. They are solid in consistency, but as a rule resonant on percussion. Some are tender, whilst others are insensitive. Tumours of the fixed portion of the intestine are as a rule immobile, but in a part of the gut possessed of a mesentery they present the greatest mobility. These mobile tumours may be fixed by adhesions in the most abnormal positions. Tumours of the intestine frequently disappear and reappear. They are simulated by masses of fæces, by external tumours adherent to the gut, and by masses of intestines adherent around the growth.

When the tumour ulcerates and fungates, blood, pus, mucus, and occasionally fragments of growth, may be found in the dejecta.

Blood is present in the motions in 20 to 25 per cent. of cases, according to R. de Bovis. Except in the rare villous form of cancer, it is not large in amount. Sometimes it appears as streaks on the fæces; in other cases it is mixed; whilst in very rare instances large hæmorrhages may occur.

Pus is found in small quantities when the growth ulcerates. It may, of course, be found in any other form of ulceration or abscess formation.

Mucus is common in cancer of the colon. It is described by the patient as slime, and is due to the catarrhal condition of the bowel above the growth.

Extension of the Cancerous Process to the Peritoneum and Omentum.—In a minority of cases this invasion is so extensive as to entirely overshadow the primary growth. The signs are those of ascites, with the presence of nodular and irregular tumours in various parts of the abdomen. The most characteristic of these is an elongated nodular mass transversely placed in the umbilical region, and formed of the rolled-up omentum infiltrated by growth. In such cases, even if signs of stenosis of the intestine exist, no certain diagnosis of cancer of the intestine can be made, for the gut may be obstructed by a secondary process of matting and infiltration.

Metastases in the Liver and Lung.—In rare cases these secondary deposits in the liver and lungs have been the most prominent signs of the disease, and intestinal obstruction has not appeared until near the termination of the case.

(*b*) *Chronic and Progressive Stenosis of the Intestine due to Cancer.*—In nearly all cases of cancer of the intestine the symptoms are those of a progressive stenosis. In practice the only common causes of chronic intestinal obstruction after forty years of age are fæcal accumulation and cancer of the colon, and the diagnosis therefore rests between these two conditions.

Several clinical types of cancerous stricture of the bowel exist.

In most cases the signs of stenosis appear in a previously healthy subject, and it is only as the case progresses that the cause can be ascertained.

In another and smaller class of cases the symptoms due directly to the growth have been present for some months before the bowels begin to be obstructed.

Finally, the symptoms of stenosis due to cancer may be grafted on those due to fæcal accumulation, perhaps with stercoral ulceration, which has produced the growth by chronic irritation.

1. *Constipation* is in most cases the first symptom of stenosis of the intestine. It is irregular at first, but progressive, so that purgatives and enemata become necessary. Purgatives at first give great relief, but after a time they cause severe pain, although they act; finally they precipitate an acute attack of intestinal obstruction. Constipation coming on after forty years of age in a previously healthy man should always suggest the possibility of cancer being present. The period during which the bowels may be closed extends to days, weeks, or even months. These periods of constipation are terminated by attacks of diarrhœa, either spontaneous in origin or as the result of drugs, and thus the alternation of constipation and diarrhœa is established. In some cases the growth ulcerates after a time, the stenosis is relieved, and the constipation ceases; but the stools will contain blood, pus, and mucus. In some cases, especially when the growth is in the lower colon, persistent irritative diarrhœa may be present.

Tenesmus is also frequently present under such circumstances. The motions may alter in shape and become small in calibre, flattened, or like the dung of rabbits or sheep. These changes are due to spasmodic contraction of the sphincter ani, excited by the irritation of the growth, which is usually in the lower colon.

2. *Attacks of Colic.*—Presently the patient begins to suffer from attacks of colic, during which he vomits and his bowels are closed, whilst his abdomen becomes distended and wind rumbles about it. These attacks sometimes amount to agony, but as a rule the pain is moderate. It is sometimes correctly localized at the stenosis, but often it is diffused and radiates widely over the body.

3. *Vomiting* is not a well-marked symptom in chronic obstruction due to cancer of the intestine. It occurs at irregular intervals, and usually after taking solid food. Frequently it is only one feature of an attack of colic.

4. *Contracting Coils of Intestine* are often seen through the abdominal wall. The coils of intestine rise up and harden during the attack of colic. Normal peristalsis may be observed when the abdominal wall is greatly emaciated, but the movements are slow, and the coils are not distended and do not harden as they contract. In the colic of enteritis and lead-poisoning the coils of intestine are not visible. When this sign is definitely present, there is no doubt that an organic obstruction is present, and it is very uncommon in fæcal accumulation. The position of the growth may be determined with some degree of probability by ascertaining whether the contracting coils are large or small intestine, and in which part of the abdomen the waves terminate. Small intestine is centrally placed, assumes the well-known ladder pattern, contracts quickly and vigorously, and is as a rule less than 3 inches in diameter. Large intestine often bulges the flank on one or both sides, and is broader and contracts more sluggishly. It must be remembered, however, that when the intestine has become exhausted just above the cancer, the waves of peristalsis may stop some distance above it.

5. *Meteorism and Distension.*—When obstruction is well established, the abdomen becomes distended. As a rule the distension is greater the lower down the canal the cancerous stricture is placed; and as cancers are usually in the sigmoid flexure or rectum, distension is a common feature of these cases. When, however, the cancer is in the duodenum or upper jejunum, intestinal distension may be absent; but the stomach may be distended even below the umbilicus. Local distension is sometimes seen. Thus, the cæcum and ascending colon may be clearly enlarged, and this would indicate a cancer at the hepatic flexure. Or the right flank may be bulged out, whilst the transverse colon can be clearly defined, which would indicate one at the splenic

flexure. Finally, when both flanks curve outwards and the left iliac region is distended, the growth is probably in the sigmoid flexure or rectum.

Nothnagel has drawn attention to the fact that distended colon gives a hyperresonant note in the corresponding costo-vertebral angle, and that this sign may be utilized to locate the obstruction, especially if it be obtained on one side only. When the whole abdomen is evenly distended, very little can be deduced, except that the obstruction is probably in the lower colon.

When the colon is enormously distended and filled with fluid fæces, the condition of the abdomen may closely simulate that of ascites. The flanks are bulging, dull on percussion, and there is a sense of fluctuation on palpation. Succussion can, however, nearly always be obtained when the fluid is within the intestine.

The symptoms of acute intestinal obstruction are present in cancer of the intestine.

In the majority of cases the symptoms of acute intestinal obstruction supervene on those of chronic stenosis, and are due to acute dilatation of the colon or cæcum. It has already been pointed out, however, that compensatory hypertrophy may be so complete that practically no symptoms precede those of complete and absolute obstruction. The causes which lead to the transition from chronic stenosis to absolute obstruction have already been detailed. A foreign body may become impacted. A distended coil of gut may be twisted or kinked, or the muscular wall of the intestine may become exhausted after the administration of a violent purge. Peritonitis frequently plays an important part in the acute termination of these cases. Acute dilatation of the colon is very often, and that of the cæcum nearly always, accompanied by general peritonitis due to stercoral ulcers. The formation of localized abscesses around a cancer may produce the final and complete obstruction, or a perforation of the colon may lead to peritonitis, and so to paralysis of the intestine and absolute occlusion. Under any of these circumstances the symptoms suddenly become acute. Continuous pain is experienced in the abdomen, marked at intervals by severe attacks of colic. Vomiting becomes frequent and urgent, and after a time is fæcal in character. The bowels are entirely closed, so that neither fæces nor flatus are passed, and turpentine enemata are returned without result. The abdomen rapidly becomes distended, very frequently to such a degree that separate coils cannot be distinguished. A careful examination will, however, often reveal contracting coils of gut, and this is a sure sign that a chronic stenosis has preceded the acute attack, even should the history fail to indicate this.

When peritonitis complicates the acute attack, the abdomen is tender and rigid, and there is as a rule some pyrexia. If a perforation has occurred, the abdomen is often evenly distended and uniformly tympanitic ; and when an incision is made into it, a large quantity of gas and fæcal matter escape.

Collapse is seldom as well marked as in such forms of acute obstruction due to bands. It is late in its appearance, and is due to toxæmia resulting from absorption of the products of decomposition from the foul intestinal contents.

The Localization of Cancer of the Intestine in the Large or Small Intestine.—The chief means of distinguishing between carcinoma in the large and small intestine will be briefly stated here. A cancer of the small intestine is latent for a longer time, because the contents are fluid. When the symptoms commence, the pain and vomiting are as a rule more severe and persistent when the growth is in the small intestine, and the pain is often found to be related to the taking of food. Constipation is a much more prominent symptom of growth in the colon, and blood and mucus are seldom present in the fæces in a case of carcinoma of the small intestine. Tenesmus, ballooning of the rectum, and fæces of small calibre, as a rule point to a growth in the lower part of the colon. In a carcinoma of the small intestine the abdominal distension is, generally speaking, centrally placed in the abdomen. When the colon is distended, one or both flanks bulge outward. When distended coils of intestine can be seen, and especially when they contract, it may be possible to identify them anatomically. Coils of small intestine are smaller, exceedingly tortuous, or arranged in a "ladder pattern," and they contract more rapidly and vigorously.

The Diagnosis of Cancerous Stricture of the Intestine.—Carcinoma of the intestine has in most cases to be distinguished from *fæcal accumulation and coprostasis.* In the latter condition there is as a rule a long history of constipation, and repeated enemata bring away increasing quantities of hard, compacted scybala. Moreover, large masses of fæces may be felt in the rectum, and contracting coils of colon are seldom seen in coprostasis. When, however, chronic constipation and stercoral ulceration have preceded and produced carcinoma, the diagnosis is exceedingly difficult.

Chronic ileo-cæcal tuberculosis may closely simulate carcinoma, especially when it ulcerates, and blood and pus appear in the motions. The presence of tubercle elsewhere and the longer history of the cases may assist in a diagnosis, but as a rule it is impossible to distinguish these conditions.

Leube has called attention to a condition of *chronic sigmoiditis* in which the sigmoid flexure is greatly thickened, and forms an elongated tumour in the left iliac region. Such cases are rare, and are scarcely capable of diagnosis.

Foreign bodies have also been mistaken for carcinoma of the intestine.

Tumours of neighbouring organs, such as the stomach, gall-bladder, uterus, and ovaries, may be mistaken for cancer of the intestine when they invade the bowel. The history and associated symptoms of such cases will as a rule enable them to be distinguished.

The Treatment of Carcinoma of the Intestine.—When the growth is inoperable and the symptoms of obstruction are slight, much may be done to alleviate the patient's condition. His food may be so regulated as to leave only a small residue. His attention should be directed to its complete mastication; and should his teeth be defective, they should be attended to by a dentist, or his food may be thoroughly minced before he eats it.

Intestinal decomposition and flatulence may be controlled by such drugs as minute doses of calomel, β-naphthol, and salol.

When definite symptoms of obstruction are present, the intestine should be opened above the carcinoma, or in certain cases it may be possible to establish an anastomosis between the intestine above and below the growth.

The possibility of excision of the growth is a surgical matter, but it should always be postponed to a second operation when distension and obstruction are present.

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(ii.) **Cicatricial Strictures of the Intestine** are the result of—(1) The contraction of scars produced by healing ulcers, or (2) the loss of tissue due to limited gangrene or sloughing; (3) in the case of syphilitic strictures and in the hyperplastic form of tuberculosis, the narrowing of the lumen of the intestine is largely due to the deposition of dense masses of connective tissue in the wall of the intestine.

As a rule the scar is annular and surrounds the gut, and in the majority of cases bending, distortion, adhesions, or folds of mucous membrane and polypi, assist in the production of intestinal obstruction due to strictures.

Incidence.—Cicatricial stricture of the intestine is an exceedingly rare cause of intestinal obstruction if strictures of the rectum are excluded, as it is usual to do. This is remarkable when the frequency and extent of many varieties of ulceration is taken into account. The surgical registers of the London Hospital contain but thirteen examples among 669 consecutive cases of intestinal obstruction in thirteen years.

Etiology.—Cicatricial strictures may be divided into—(A) Those due to some form of ulceration; (B) those which follow abdominal trauma; (C) those which result from strangulated herniæ. Those simple strictures which follow the spontaneous elimination of an intussusception are fully dealt with under that heading.

The thirteen cases at the London Hospital were distributed as follows:

(A) Ulcerative, 9—that is, tubercular, 4; syphilitic, 2; duodenal ulcer, 1; undetermined, 2. (B) Traumatic, 3. (C) Strangulated hernia, 1.

Nothnagel states that for practical purposes tubercular and stercoral ulceration are the only common causes of cicatricial stricture. A few cases are, however, due to syphilitic ulcers, round ulcers of the duodenum, dysenteric and catarrhal follicular forms of ulceration, and typhoid ulcers.

(A) **Strictures due to Ulceration**—1. *Tuberculous Strictures of the Intestine.*—The tuberculous infection may attack either—(*a*) The mucous membrane, (*b*) the submucous tissue, or (*c*) the muscular coat and subserous layer.

(*a*) *Tuberculosis of the Mucous Membrane.*—In this form of tuberculosis the process commences in the lymphoid tissue of the Peyer's patches or solitary follicles. Miliary tubercles are formed, and the patch breaks down into a ragged ulcer with overhanging edges, which tends to spread transversely around the gut. If the process is chronic, some of the ulcers may heal, and a moderate degree of stricture is not uncommon, but actual stenosis producing symptoms of obstruction is rare. Tuberculous ulcers are more common in the ileum, and diminish in frequency towards the stomach. Strictures produced by them are often multiple (Handford, Voehts, Nothnagel, Rolleston, Mayo Robson); as many as seven or eight have been recorded in one case.

(*b*) *Tuberculosis of the Submucous Layer.*—In this form there is a round-celled infiltration interspersed with giant-cells in the submucous tissues. The mucous membrane itself is not ulcerated. According to Tuffier, as the lesion heals and contracts it produces diaphragm-like strictures.

(*c*) *Tuberculosis of the Muscular and Subserous Coats* produces the rare chronic hyperplastic tuberculous tumours of the intestine. In these cases the muscular coat is greatly thickened owing to the deposition of dense fibrous tissue, but the chief deposit of this tissue is beneath the serous membrane, which may reach a thickness of from ½ to 1 inch. Fat and giant-cells are found on section, and a few tubercle bacilli. The lumen of the bowel is reduced to a narrow channel, which may or may not be ulcerated. The extreme hyperplasia is due either to a mixed infection or an attenuated form of the tubercle bacillus. Conrath collected eighty-five cases in 1898, of which the majority were in the ileo-cæcal region (tumour-like tuberculosis of the cæcum), but it also occurs in the small intestine and other parts of the colon. Mr. F. S. Kidd has recently reported three cases in the sigmoid flexure.

These strictures have frequently been mistaken for carcinoma even after excision, but it is said that the limits are more diffuse; the infiltration disappears gradually into the neighbouring intestine, and symptoms of stenosis appear later.

In all forms of tuberculous stricture the neighbouring lymphatic glands are affected, and the mass is adherent to the omentum or adjacent organs.

The chief points in the diagnosis of a tuberculous stricture are the presence of phthisis and of tubercle bacilli in the fæces; but both are often absent, and, as Lartigan has pointed out, the infection of the intestine may be primary. Bernay has collected 70 cases of tuberculous stricture. Of these only 8 were of the hypertrophic variety; 45 were submitted to operation, and 37 recovered. In most cases the mass was resected, but in some it was excluded by a lateral anastomosis.

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2. *Stercoral or Decubital Ulcers* are due to the pressure of hard masses of fæces. They are therefore found most frequently in the sigmoid flexure, rectum, cæcum, and the hepatic and splenic flexures of the colon. Grawitz and Nothnagel believe that they are a frequent cause of simple stricture, and are often mistaken for syphilitic and carcinomatous stenoses. The latter holds that cancer frequently originates in the annular scar left by a stercoral ulcer, and he has recorded such a case and quotes another by Treves. The condition must, however, be exceedingly rare, as no case of stenosis of the intestine due to stercoral ulceration has been recorded at the London Hospital in thirteen years.

3. *Duodenal Ulcer.*—In consequence of the broad diameter of the duodenum and the fluid character of its contents, it is very exceptional for the scar of an ulcer to cause stenosis. Many varieties of ulceration occur in this situation, but only the simple ulcer, which is equivalent to the peptic ulcer in the stomach, appears to produce obstruction. This appears to be due to the infective character of this form of ulcer both in the stomach and duodenum, a form which produces, in my experience, a contact ulcer opposite to it in practically every case. A pair of such

ulcers near the lesser curve of the stomach frequently fuse as they spread, and form a single horseshoe or crescent ulcer, which, as it cicatrizes and contracts, produces an hour-glass stomach. In a similar way I have seen duodenal ulcers nearly surrounding the lumen of the intestine. A stricture of the duodenum due to a simple peptic ulcer was under the care of Sir Frederick Treves in the London Hospital in 1896. The man, who was thirty-two years old, had suffered for two and a half years with symptoms of pyloric stenosis. The stricture was dilated with the index-finger (Loreta's operation), and the symptoms relieved, but he died of profuse hæmatemesis a month later. Duodenal ulcers are as a rule situated in the first part of the duodenum, above the biliary papilla. In rarer instances they involve that papilla and produce persistent jaundice, whilst in yet other cases they are near the duodeno-jejunal flexure. Should a stricture result in the latter situation, bile is present in large quantities in the vomit. Hochhaus has reported three cases of duodenal stenosis due to gallstones; one involved the pylorus and duodenum, one was in the first part of the duodenum, and one near the jejunum. Obstruction in these cases, however, appears to have been due rather to constricting adhesions than to true stricture.

4. *Syphilitic Strictures of the Intestine.*—Syphilitic ulceration and stricture is common in the rectum, especially in women; but the cases recorded in the small intestine and colon are often doubtful. Two such cases have been in the London Hospital. The first was a man aged thirty-four, under the care of Mr. Mansell Moullin in 1894. A simple stricture was found in the lower ileum, for which lateral anastomosis was performed, but the patient did not survive. The registrar reported that the specimen appeared to be syphilitic. The second case was under the care of Mr. James Sherren in 1903. The patient was a man aged thirty. A gummatous mass was found in the jejunum, and was resected with 10 inches of gut. The patient recovered. Syphilitic ulcers are said to occur both in the congenital and acquired forms of the disease. Apart from the rectum, they nearly always occur in the tertiary stage. Gummata form in the lymphatic follicles, in the mucous membrane, or in the submucous layer, and these break down into ulcers, which are transversely disposed and have a hard base and a greyish-white margin. The ulcers may be single or multiple. They are said to be more common in the jejunum.

5. *Typhoid Ulcer* practically never leads to stenosis of the

intestine. Only two undoubted cases have been recorded. Nothnagel cites one by Klob, and a later one by Hochenegg. The reasons that have been offered for this strange immunity from stricture are that the ulcers are often small and disposed along the axis of the gut, and that when the ulceration is deep and extensive the patient does not survive.

6. *Dysenteric Ulceration* is scarcely ever a cause of stenosis of the intestine. Woodward reported that during the American War of Secession there occurred among the United States troops 287,522 cases of dysentery, and of these 28,451 were of the chronic form; 9,431 died, and 3,855 of these were chronic cases, yet not a single instance of stricture of the intestine was found.

(B) **Strictures after Injury.**—Resection of the intestine, followed by anastomosis with Murphy's button, is responsible for many of these. Mr. T. H. Openshaw resected a carcinoma of the sigmoid flexure in 1902, and united the ends with a Murphy's button in the London Hospital. In 1906 the woman, who was aged sixty-one, returned with a fibrous stricture over which a fistula had formed. Stricture may, however, follow the method of suture, and even that of lateral anastomosis.

An abdominal injury, such as a kick, a blow, or the passage of a cart-wheel, may partially rupture the bowel, and yet the patient may recover. Stricture is then liable to follow in from one to four months. Treves has collected six cases, and I have had two under my care. One, a little girl aged three, was run over in August, 1905. The mark of the wheel crossed her abdomen, and she was profoundly collapsed, but she recovered. In December, 1905, she was readmitted to the London Hospital with complete intestinal obstruction of eight days' duration. The ileum was found strictured and adherent to the abdominal wall at two points about 2 inches apart, and a large aperture was present in the mesentery. The intestine ruptured whilst it was being separated, and a Paul's tube was inserted. The patient did not survive. The second case was that of an ostler, aged forty, who had been kicked in the pit of the stomach by a horse five months previously. He vomited and was laid up several days, and had suffered from abdominal pain ever since. He was admitted to the London Hospital in July, 1905, suffering from colic and contracting coils of small intestine. A coil of jejunum was found to be kinked, with a scar crossing it obliquely and covered by adherent omentum. Lateral anastomosis was performed, and he made a good recovery.

(C) **Strictures after Strangulated Hernia.**—Only one such case has occurred in the London Hospital in thirteen years. This patient was a woman, aged forty-two, under the care of Mr. F. S. Eve in 1903. She had been operated on for a left strangulated femoral hernia some months previously, and had suffered from symptoms of stenosis of the small intestine for six weeks. Two strictures were found in the ileum, a few inches apart, and the mesentery was puckered between them. They were resected together. The patient did not survive. Sir Frederick Treves has collected eleven such cases. In nine the ileum was involved, and in two the jejunum. One case was double, the rest were for the most part narrow annular strictures; but in one the bowel was contracted for 1½ inches.

The symptoms of stenosis appear in from a few days to some years after an inguinal or femoral hernia has been reduced by taxis or operation. It is probable that the piece of intestine which has been injured by the neck of the sac undergoes a local annular gangrene of its mucous and submucous layers. The repair of this loss of tissue produces the stricture.

The Anatomy of Cicatricial Strictures.—Nothnagel and Treves state that cicatricial stricture is much more common in the colon than in the small intestine, and the latter gives the proportion as 6 to 1. They include, however, strictures of the rectum, where syphilitic, stercoral, dysenteric, and traumatic ulcerations are common. Moreover, contraction of the lumen without symptoms is apparently counted as a stricture. Of the thirteen cases at the London Hospital, four were in the sigmoid flexure and nine in the small intestine.

The length of the stricture varies within wide limits. As a rule it is short, and often appears as though a string had been tied round the intestine. Syphilitic and tubercular strictures frequently assume the form of tumours some two or three inches long. Where extensive ulceration of the colon has produced stenosis, the intestine may be reduced to a rigid narrow tube for a considerable distance.

The contraction of the intestinal lumen necessary to produce symptoms depends largely upon the site of the stricture. In the small intestine, where the contents are fluid, the lumen is nearly obliterated before symptoms arise, unless a foreign body becomes impacted. In the rectum and sigmoid flexure, however, where the intestinal contents are normally solid, a moderate degree of narrowing will produce obstruction.

A cicatricial stricture is as a rule adherent to neighbouring structure, and surrounded by peritoneal exudate and adhesions.

The stricture itself is formed of connective tissue, which is usually pigmented, and a round-celled infiltration is often present. In the case of tubercular strictures, giant-cells and tubercular bacilli are usually found, and in that of syphilitic strictures peri- and endarteritis are the features of the section.

The changes above the stricture have been already dealt with in the General Pathology of Intestinal Obstruction.

The Symptoms and Diagnosis.—The symptoms of a cicatricial stricture are those of stenosis, and need not again be given.

In the diagnosis of an innocent stricture of the intestine from one due to cancer, the main points to which attention should be paid are the long duration of the case without the production of cachexia. The history of previous enteritis and ulceration, trauma, or a strangulated hernia, may often be obtained.

Fæcal accumulation seldom gives rise to contracting coils of intestine, and this is the chief point in its diagnosis from cancerous stricture.

It must, however, be remembered that fæcal accumulation produces stercoral ulcers, and these, when they heal, annular strictures, which, according to Nothnagel, frequently become malignant, so that the symptoms of these conditions pass imperceptibly into one another, and it is often impossible to make a definite diagnosis.

Treatment.—When a diagnosis has been made, the case should be transferred to the care of a surgeon. The treatment is carried out on similar lines to that of a cancerous stricture, but, for obvious reasons, lateral anastomosis is more often indicated.

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(iii.) **Congenital Occlusion and Stenosis of the Intestine.**—Congenital ano-rectal stenosis and occlusions are not included in this description.

Incidence.—There were 7 cases of congenital occlusion or stenosis of the intestine in 669 consecutive cases of all forms of obstruction at the London Hospital, and 6 further specimens have been brought together in the museum by Professor Arthur Keith. Leichtenstern states the distribution and proportion are as follows: 75 small intestine, 10 colon, 375 at the ano-rectal junction. The 13 cases at the London Hospital were distributed as follows: Jejunum, 2; connected with Meckel's diverticulum, 6; ileo-cæcal, 2 (and in one of these, in addition, the colon terminated blindly at the splenic flexure—*vide* Fig. 14); upper sigmoid, 1; lower sigmoid, 2. The walls of this narrowed portion may be normal in structure. Such a case at the recto-sigmoid junction, 1½ inches long, was under the care of my colleague, Mr. Hugh Rigby (see

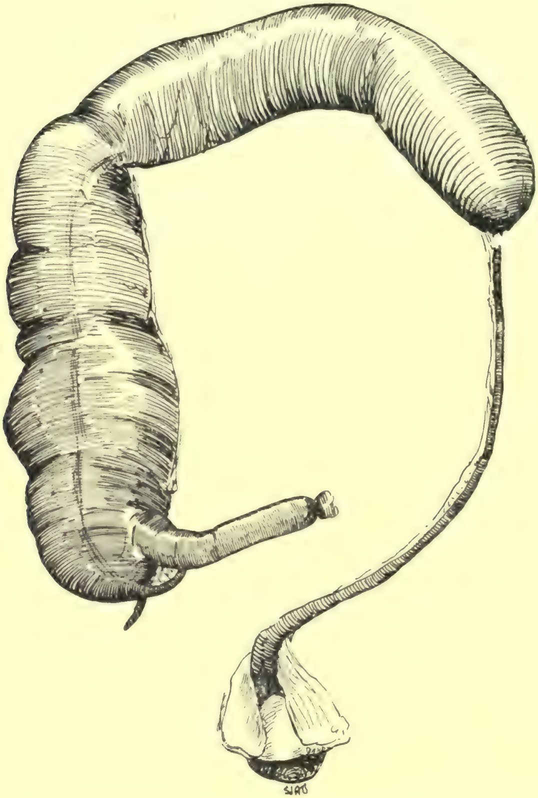

FIG. 14.—TO ILLUSTRATE A CASE OF CONGENITAL OCCLUSION OF SMALL INTESTINE IN WHICH THE COLON TERMINATED AT THE SPLENIC FLEXURE.

also Congenital Dilatation of the Colon, p. 148). The strictures in connection with Meckel's diverticulum are more often above than below that structure.

Congenital Idiopathic Dilatation — *Sex.* — *Congenital idiopathic dilatation of the colon* occurs far more frequently in male children, in the proportion of 23 males to 4 females.

Age of Onset.—The age at which the symptoms are first noticed is from the day of birth up to about the age of three months. In the case recorded by Hobbs and de Richemond,[1] the abdominal distension actually interfered with the birth of the child. In one of Hirschsprung's[2] cases the baby passed no motion for three days after birth, when the passage of a finger through the anus induced the escape of a large quantity of meconium. This child died, when two and a half months old, from distension and total inability to defecate spontaneously except after purgatives.

In some cases the onset of the symptoms is delayed until the third month, or even later.

The chief symptoms are *constipation and distension.* Constipation practically always precedes distension by a month or two. The baby simply fails to evacuate its bowel unless strongly stimulated to do so. After a few months the abdomen becomes greatly distended during these periods of constipation, and often rapidly so. When the bowels are moved in the early days of the disease, the distension disappears. This distension is very largely a matter of flatus, for when a rectal tube is passed a large quantity of wind will often escape, and the little patient be much relieved. At first the fæces are soft and normal in consistency, and are, clearly, not the cause of the complaint. As the disease advances, and fæces remain for long periods in the colon, in some cases they become scybalous and hard, but this is exceptional. When the disease has become established, the bowels are frequently not opened for periods varying from one to three weeks. After a time colitis and stercoral ulceration are produced, and attacks of diarrhœa punctuate the progress of the complaint. Far from relieving the little sufferer, these attacks are frequently attended with increased distension, severe pain, and rapid wasting, and in many cases the children have died in such attacks.

When attacks of diarrhœa have not yet occurred, the health of the child is little affected by its prolonged periods of constipation and distension, and it suffers from very little abdominal pain or discomfort.

The way in which the colon reacts to the means employed to empty it is worthy of note. There appears to be a curious sluggishness in the normal reflex action for evacuating the bowel, so that defecation is not excited by accumulation of fæces and distension of the colon.

It has already been narrated that in one case the distended colon was emptied by the simple mechanical procedure of introducing the forefinger. In many cases where great and tense distension has existed, the passage of a rectal tube has enabled a vast quantity of gas, and less often fluid fæces, to escape, so that it has been difficult to understand what hindered its evacuation before. Apparently the reflex from the lining membrane of the rectum and sigmoid is deficient, for many cases have shown an abnormal capacity for retaining one enema after another up to the number of three or four.

At long intervals the sluggish colon awakes to action, and can be seen through the thinned abdominal wall, endeavouring by slow but powerful waves of peristalsis to propel forward the huge accumulation it has permitted. The great hypertrophy of the wall of the gut is apparently acquired in consequence of these occasional efforts. The child is sometimes afflicted with severe colicky pains whilst these efforts continue, but very often they appear to cause no discomfort.

A very curious fact is that the colon will sometimes for periods of as long as a year at a time perform its functious normally, and then, without any obvious reason, once more relapse into its sluggish condition. Vomiting is rare.

The distension, as has already been said, follows the constipation in a month or two. In rare cases it has been delayed for a much greater length of time. In only one case, that of Walker and Griffith,[3] has it preceded the constipation. As age and the disease progress, it becomes greater and greater, and should the patient survive to adult life, it may attain immense dimensions. Formad's[4] case reached the age of twenty-nine, and was exhibited as a freak under the title of the Balloon Man. His girth was 86½ inches. The rapid increases of distension which are often seen are due to the formation of gas within the colon. The abdomen comes to be an enormous rounded body, around which the rest of the body and the limbs are arranged as appendices (*vide* Fig. 15). The margins of the lower ribs are everted, and the subcostal angle is permanently opened out. The thoracic viscera are pushed high up into the thorax. Dyspnœa is not rare, and the veins coursing up the abdomen are usually dilated. Œdema of the legs, scrotum, and lower abdominal wall, has been recorded.

The outlines of the huge colon can as a rule be seen through the abdominal wall, especially when slowly contracting. The note on percussion is as a rule tympanitic throughout, though fæcal tumours can often be felt in the later stages of the disease. According to Dr. Griffith,[5] fæces are seldom found in the rectum, as this part of the great gut is the least often involved.

Prognosis.—This disease almost invariably ends fatally, and but few of the patients attain to adult life. The figures given by Dr. Griffith[5] are as follows :

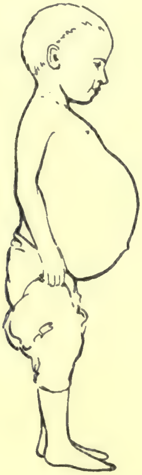

FIG. 15.—FROM CONGENITAL IDIOPATHIC DILATATION OF THE COLON. J. P. CROZIER GRIFFITH, M.D.

> Of twenty-four cases, eighteen are known to have died of the disease ; but three of these attained to adult life ;
> Fifteen cases died under the age of fourteen years ;
> Eleven cases died under the age of five years ;
> Six cases died under the age of three years.

Of the six cases whose death is not recorded, four disappeared from observation. Dr. Cheatle's[6] case recovered after medical treatment, and one of Dr. Osler's[7] as a result of the formation of an artificial anus in the distended sigmoid flexure.

Morbid Anatomy.—The following morbid anatomy is based upon the results found at eighteen post-mortems and one laparotomy.

The morbid changes were strictly limited to the distended colon and the consequences of this distension. In none of these cases was any stricture, bending, volvulus, or other organic cause, found which could account for the condition.

The colon was found distended throughout more or less of its extent in every case, and, with only one exception, its wall was hypertrophied to a thickness of from $\frac{1}{3}$ to $\frac{1}{2}$ inch. In several cases it is distinctly stated that both circular and longitudinal fibres shared in this thickening. In many of the cases the colon was ulcerated, but this must be regarded as a secondary and usually late result of the fæcal accumulation. Ulceration is, moreover, the commonest cause of death, and is therefore very frequently seen at autopsies on such cases. Some dispute has arisen as to whether this disease is essentially one of the sigmoid flexure, or whether it more often involves the colon above it as well. Dr. Griffith[5] is of the latter opinion, which he bases on the following distribution of distension in the above nineteen cases :

In two cases the sigmoid flexure alone was distended.
In two cases the sigmoid flexure was greatly distended, and the rest of the colon in a slight degree.
In fourteen cases the sigmoid flexure was greatly distended, and the rest of the colon as much, or even more, dilated.
In one case the distension was entirely above the region of the sigmoid flexure.

It must, however, be remembered that these are the results of the autopsies after the last of many attacks of intestinal obstruction, and that death occurred in nearly every case because these last attacks could not be relieved. It is therefore not surprising that the upper part of the colon was distended with fluid fæces and with gas. There is little doubt, however, that the condition is nearly always primarily one of the sigmoid flexure, and that its effects are gradually propagated back along the colon as time goes on. The sigmoid flexure of the Balloon Man was 30 inches in circumference.

The rectum is usually contracted and normal. In a few cases the distension has extended into its upper part, and very rarely it has been involved by distension and hypertrophy as far as the anus.

The other abdominal organs are greatly displaced by the huge distension of the lower part of the colon. The small intestines are forced up into the right hypochondrium. The liver has been found rotated so that it lay entirely to the right of the middle line, with its long axis vertically placed. The dome of the diaphragm is greatly raised, especially on the left side, where it may reach the level of the second intercostal space or the third rib. The lungs are forced up into the upper part of the pleuræ, and their bases are compressed. The heart may be found situated behind the manubrium sterni.

Pathology.—In a condition the cause of which has led to so much dispute, it would seem well to start a discussion of the pathology by a review of the physiology of the organ involved, and to follow this by an enumeration of the chief facts about the disease which seem to bear on its cause.

The function of the descending colon and sigmoid flexure is to extract the fluid from the fæces, and to store them until a convenient occasion arrives for their evacuation. It is very possible that a functional sphincter separates the sigmoid flexure from the rectum. The sigmoid flexure has long periods of rest during which it receives and stores the solid fæcal matter, and short intervals of activity in which it empties its contents into the rectum. In the normal individual the desire to defecate is excited, and the rectum evacuates the fæcal matter.

The stimulus to these reflex actions is, apparently, the mechanical contact and irritation of the mucous membrane by fæcal matter, and the distension of the colon, especially by flatus. The facts about congenital idiopathic dilatation of the colon which are relevant to its etiology are as follows: The baby from its birth shows no desire to defecate. In most cases this would appear to be due to the fact that the fæcal matter does not automatically pass from the sigmoid flexure into the rectum.

Enemata are frequently retained in succession, and the fatal issue is usually due to their total failure to excite the colon to contract. The passage of a rectal tube past the rectum into the sigmoid flexure will often lead to the escape of flatus and fluid fæces, which were obviously under considerable pressure. In the early stages of the disease the bowels may act normally for long periods.

The dilated coil may be seen undergoing slow and powerful peristaltic contractions after it has become distended during a long period of constipation. The wall of the dilated coil is practically always hugely hypertrophied. The rectum is in the great majority of cases normal. The cause of the disease would then appear to be *an exceedingly lethargic reflex* for the evacuation of the sigmoid flexure, and less often of the rectum.

A normal quantity of fæces or flatus does not excite the sigmoid flexure to reflex expulsion. It is only when the bowel has become hugely distended by the constant addition of fæcal matter from above that the reflex is tardily excited. The bowel has then to deal with a very large mass of fæcal matter, which is usually quite beyond its powers, and it requires the stimulus and assistance of copious enemata. These occasional but violent efforts lead to the great hypertrophy of the bowel wall. It must be remembered that the power required to empty a circular cavity varies as the cube of its radius.

If the colon did not become hypertrophied in this proportion, it would fail to propel its contents at all, as, indeed, it ultimately does. Nor can this hypertrophy be pointed to as evidence of obstruction beyond the sigmoid flexure. It is a compensation for the increasing mass to be moved, and the ever-enlarging area over which the muscular power of the colon must be spread. Many surgeons believe that there is always *a mechanical cause* for congenital dilatation of the colon, and that the name "idiopathic" is a misnomer. Undoubtedly cases of congenital dilatation of the colon are due to congenital strictures and other malformations, and without the definite evidence of an autopsy or a laparotomy it is not possible to be quite certain that such a mechanical obstruction is not present.

In nineteen cases, however, the absence of such mechanical cause has been definitely excluded, and in two of the cases which occurred at the London Hospital a stricture, or kink, or volvulus, was definitely searched for, and was not found. There is therefore no doubt that, apart from cases of dilatation due to a mechanical cause, there are others in which no such cause is present.

It is generally recognized that the rapid formation of large quantities of gas in the alimentary canal is frequently due to vascular causes. The rapid distension which is so marked a symptom of this disease is often very largely gaseous. For this reason it has been suggested that this disease may have *a vascular origin*, but, as far as I am aware, no facts or observations have been brought forward to support this theory.

The *Diagnosis* of congenital idiopathic dilatation of the colon is readily made from the symptoms. As has already been stated, an autopsy or a laparotomy alone can enable us to be quite certain that no congenital deformity of the gut is present.

Treatment: 1. *Curative.*—In only three cases has treatment been successful. Dr. Cheadle's[6] case was cured by mild laxatives and tonics, and daily motions were obtained, but the information is not complete enough to say that this was a permanent cure.

In Dr. Osler's[7] case an artificial anus was established in the dilated coil, and the symptoms had not returned several years later, when the child was lost sight of.

In Dr. Griffith's[5] case, Dr. John Ashhurst, junior, established an artificial anus just above the cæcum, but the patient did not survive.

In a case of congenital dilatation of the colon due to a congenital narrowing of the rectum, Sir Frederick Treves[8] successfully excised the whole of the dilated area with the rectum, and brought the end of the normal colon out of the anus.

In my own case I divided the ileum and anastomosed the proximal end as low down in the rectum as possible. This case had not relapsed after two years.

CASE.—Congenital idiopathic dilatation of the colon. Ileo-colostomy; cure.

Bernard D., aged sixteen years, was sent up to me at the London Hospital by Dr. Eric Battiscombe. He had suffered from severe constipation and distension since his birth. For periods of as long as three weeks he had passed no motion at all, and had become greatly distended. At the end of these periods massage and enemata had brought away huge quantities of fæces, on some occasions as much as would fill three "chambers." During long intervals the bowels had acted naturally, and regular daily evacuations had been obtained, on one such occasion, for a period of twelve months. Latterly the periods of constipation had become more frequent and severe.

The boy was small for his age and poorly developed, but intelligent. The abdomen was very distended; the costal margins were expanded and everted, and the subcostal angle was very obtuse. A large mass could be felt below and to the right of the umbilicus, reaching as high as the right costal margin above, and crossing the middle line above the pubes below. Slow waves of peristalsis passed down this mass from above, and during these the boy suffered from colicky pain. The rectum was full of hard scybalous masses. Under an anæsthetic the anus was dilated and the rectum cleared of the hard scybala by lavage and the finger. A copious flood of fæces of the consistency of porridge then poured out and filled several vessels. When the bowel had been thoroughly emptied, it was decided to anastomose the ileum as low down in the colon as possible.

The Operation.—The abdomen was opened in the middle line below the umbilicus. The sigmoid flexure and rectum were found enormously dilated, and their walls about $\frac{1}{3}$ inch thick. The descending colon was normal. The ileum was divided some 6 inches from the cæcum and the ends invaginated. The proximal end was then united by lateral anastomosis with the rectum as low down as possible, but in a part which was dilated and hypertrophied. The abdominal wall was closed in layers.

The patient made an uninterrupted recovery, and while he was in hospital, and for two years afterwards whilst under observation, he passed a motion naturally.

2. *Palliative.*—The child is usually brought for treatment of an attack of constipation and distension which has already lasted several days. The immediate indication is to empty the colon as rapidly as possible. Purgatives are contra-indicated unless other means entirely fail, for they increase the load and distension of the already exhausted colon.

In many cases the high rectal tube has effected the escape of a very large quantity of flatus and fluid fæces, and has temporarily relieved the distension. The solid fæces can be removed in nearly every case by the persevering use of soap-and-water or turpentine enemata. It is, however, very disconcerting when they are retained one after the other, but the high rectal tube will usually secure their return. Puncture of the dilated coil through the abdominal wall with a very fine trocar has been resorted to where all other means have failed. When the colon is empty, laxatives, especially aloes, combined with an enema every day or two, will usually prevent a re-accumulation. Massage, electricity, tonics, and attention to the general health, should be employed when the attack has passed.

The success of Dr. Cheadle's case, in which such simple means were employed, should encourage their persevering trial.

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3. Causes outside the Intestine.

(i.) **Anomalous Forms of Obstruction due to Peritoneal Adhesions.**—Under this heading are brought together a very heterogeneous group of cases, which have, however, this in common, that in all the lumen of the intestine is obstructed by some arrangement of peritoneal adhesions which does not interfere with the vessels or produce strangulation.

Etiology.—The adhesions are the result of an adhesive peritonitis, and this is due in most cases to appendicitis, diseases of the pelvic organs, tubercular peritonitis, and cancer of the intestine. Intestinal obstruction is not rare as a complication after operations on these conditions, especially where much gauze has been left or the intestine has been extensively handled.

Incidence.—Thirty-eight cases of intestinal obstruction due to adhesions have occurred at the London Hospital in thirteen years among 669 cases of all varieties. Of these, thirty-four were men and fourteen women. Twenty-six died, giving a mortality of 68·4 per cent.

Eight further cases of kinking occurred in the same series. Five were men and three women. Six died, making a mortality of 75 per cent.

Anatomy.—The results of adhesions may be divided into—(1) Kinking; (2) bending; (3) looping; (4) constriction; (5) matting; (6) shrinking of the mesentery.

1. *Kinking of the Intestine.*—When traction is exerted on an adhesion, band, or diverticulum, which is adherent to the margin of the intestine, the bowel is kinked and its lumen is closed. The sudden traction may be due to distension of the coil of intestine or to some change in its position, or the other end of the adhesion may be attached to some hollow cavity which has been emptied—such, for instance, as the uterus, an ovarian cyst, or an abscess cavity. The symptoms of kinking are as a rule those of acute intestinal obstruction, but they are not so severe and sudden in their onset as in strangulation by bands.

2. *Bending.*—In this variety of obstruction due to adhesions, the bowel is bent once or several times, and is fixed in that position. The small or large intestine may be the site of bending, and the condition may follow any of those causes of peritonitis which have been narrated, such as hernia, appendicitis, salpingitis, or operations on these.

The mechanical production of obstruction in bending is composite. The bowel is frequently kinked or constricted at one or more of the bends, and in addition more or less of the intestinal wall is immobilized by the adhesions.

As an example of a single bend, I might mention a case of acute intestinal obstruction which followed ten days after an ectopic gestation had been enucleated from the broad ligament. I found a coil of ileum adherent and acutely bent at the bottom of the pelvis. On freeing it, the contents gurgled past into the contracted gut below. The following is a good example of multiple bends: A woman reduced an umbilical hernia, and was seized with symptoms of acute intestinal obstruction. I opened her abdomen, and found the middle of the transverse colon bent no less than six times, and fixed by matted omentum. I separated the adhesions and removed the omentum. Her bowels were freely opened next day, but she did not recover.

3. *Looping.*—One part of the intestine may be adherent to an adjacent part, so that the bowel between them is converted into a fixed loop. The causes of this condition are glandular disease in the mesentery, the local peritonitis over an intestinal ulcer, or constriction and inflammation in a hernial sac. The loop may be open, in which case another coil of gut may pass through it. On the other hand, the aperture formed by the loop may be closed by adhesions. In this case obstruction is caused either by kinking or bending at the apex of the loop, or by the adherent walls between the coils acting as a valvular septum. Loops are usually dependent in the abdomen, but in two cases I have seen them hanging from adhesions between their extremity and the upper part of the abdominal cavity. In both cases the ascending limb was enormously dilated and hypertrophied, and the descending limb narrow and contracted. In both I freed the adhesions, and in one lateral anastomosis was performed. Both cases recovered.

4. *Constriction* or external stricture is a condition in which the adhesions around a coil of intestine narrow its lumen. It has already been pointed out that in nearly all cases of internal stricture adhesions are present around the intestine, and contribute to the stenosis. As a rule a single constricting adhesion binds the coil of intestine down to the abdominal wall, but I have operated on a curious case where a coil of ileum, being almost severed by a constricting band crossing it from the mesentery, was free in the peritoneal cavity. I performed lateral anastomosis, and the woman perfectly recovered.

In many cases the adhesions are multiple and irregular in their shape and direction, and produce obstruction by their cumulative effect. Leichtenstern describes a circumscribed chronic constricting peritonitis which occurs at the hepatic, splenic, and sigmoid flexures of the colon. He regards it as a secondary result of coprostasis. Nothnagel agrees with this view, but states that stercoral ulcers are usually present.

The hepatic flexure is frequently constricted by adhesions due to gall-stones, cirrhosis of the liver, and corset liver; the splenic flexure by adhesions from perisplenitis; and the sigmoid flexure by those arising from the female genital organs.

5. *Matting.*—In this form of adhesions a greater or smaller extent of the intestines is matted together in an inextricable mass. The causes of the peritonitis are those already narrated, but when the condition is universal it is usually due to tuberculous glands, chronic sclerosing peritonitis, or cancer of the intestine. It is a matter of astonishment how much of the intestine may be matted together and bound down without producing any symptom of obstruction. Indeed, this occurs in nearly every case of local and general peritonitis which recovers. Obstruction, when it supervenes, is a composite and cumulative process consisting of kinks, bends, loops, constrictions, and often twists.

Sir Frederick Treves has pointed out that a portion of the small intestine may be matted into a definite rounded tumour, which is, however, resonant on percussion. Malcolm has recorded such a case which resembled a broad-ligament cyst, and he refers to others in which the resemblance was so close that the mass was incised or aspirated. My colleague, Mr. F. S. Eve, has recently (1907) had such a case under his care. Symptoms of chronic obstruction culminated in an acute attack. A definite rounded mass of matted small intestine was found to have undergone rotation, so that the entering and leaving coils were twisted. Mr. Eve performed lateral anastomosis, and the patient recovered.

6. *Chronic Mesenteric Peritonitis; Shrinking of the Mesentery.*—This is an insidious cicatricial process which may involve the whole peritoneum, but appears to originate in the mesenteries. These structures shrink and shrivel until the intestine is fixed firmly to the spinal column.

The process is believed by Virchow and Nothnagel to originate in the glands of the mesentery. This adenitis is due to some intestinal irritation, and is often a part of coprostasis.

Virchow states that it usually originates in the glands on either composite. The bowel is frequently kinked or constricted at one or more of the bends, and in addition more or less of the intestinal wall is immobilized by the adhesions.

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Virchow states that it usually originates in the glands on either sacculi, and by sarcoma or carcinoma of the prostate. Finally, by tumours and abscesses arising from the pelvic bones or the sacro-iliac joint. It is stated that in fæcal accumulation the obstruction is sometimes due to the descent of the loaded cæcum or sigmoid flexure into the pelvis, with consequent compression of the rectum.

The fixed portions of the colon may be compressed by retroperitoneal sarcoma or other growths; by cystic kidneys, hydronephrosis, or renal sarcoma or carcinoma; by abscesses around the kidneys, cæcum, or in the psoas sheath; or by tumours of the liver, gall-bladder, or spleen.

The small intestine may be pressed upon by mesenteric cysts, tumours, glands, or hydatids; by omental cysts and tumours; by floating spleens and pedunculated kidneys; or even by a coil of intestine becoming caught between the liver and the ribs.

The duodenum has been compressed by pancreatic cysts, abscesses, and cancer; by glands in the small omentum; and by hæmorrhage into the cellular tissue around it. The following is a good example of the latter condition. A small boy was run over across the upper abdomen. His abdomen was soft and not tender, but his stomach became greatly distended and he vomited repeatedly. I performed laparotomy, and found his duodenum was surrounded by a large blood-clot, but the peritoneum over it was intact. His abdomen was closed, and he was rectally fed for a few days until the gastric distension subsided and he was able to take food without vomiting. He made a perfect recovery. Pressure on the duodenum by the superior mesenteric vessels and nerves will be treated separately.

It is stated by Patel that the intestine above a simple compression is dilated, but not hypertrophied unless ulceration is present. Ulceration is, however, not infrequent in compression, especially in the rectum, and it may then produce diarrhœa. In a case under the care of the writer, a large hydatid, situated in the recto-vesical pouch, pressed upon the rectum. A large fæcal mass accumulated above the point of compression, and produced a stercoral ulcer some two or three inches in diameter, from which persistent diarrhœa resulted.

The *Symptoms* of compression of the intestines from without present great variety. Sir Frederick Treves collected 22 cases, and of these 12 were acute, 2 were subacute, and 8 presented chronic symptoms. The proportion of acute cases in this series is probably excessive. Leichtenstern states that it is far more frequent for symptoms of chronic stenosis to precede the symptoms of acute occlusion. The latter is due to sudden changes in the position of the tumour or its rapid growth ; to the lodgment of a foreign body at the point compressed ; to the paralysis and distension of the intestine above, or kinks or volvulus of the dilated coils of intestine.

Since strangulation does not occur, the onset even of acute symptoms is as a rule less abrupt, and the pain and collapse are less severe than in those forms of obstruction in which the veins are compressed. The tumour which is producing the symptoms can as a rule be felt, and its nature diagnosed.

Treatment.—The treatment is surgical. Where it is possible, the tumour is removed or emptied, as in the case of a pregnant uterus, an abscess, or a hydatid. Where this cannot be done, either a fistula is established above the obstruction, or an intestinal anastomosis is performed so as to short-circuit the obstruction.

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Pressure on the Transverse Part of the Duodenum by the Superior Mesenteric Vessels and Nerves.—This cause of intestinal obstruction was first described by Rokitansky in 1842. Papers followed by Kundrat and Snitzler. Albrecht reported two cases of his own, and collected the published cases up to 1899. Dr. William Ewart (1899) contributed the first paper in English, and in 1900 Robinson independently described the condition in America. The matter has been more recently referred to by Kelling (1901), Campbell Thomson (1902), Zade, Neck, Nothnagel (1905), and Finney (1906).

Incidence.—Robinson found this condition more or less marked in 15 to 20 of 560 autopsies, a proportion of from 3 to 4 per cent.

Morbid Anatomy.—The superior mesenteric artery arises from the aorta behind the pancreas, and, issuing from the lower border of that organ, crosses the duodenum transversely, where it lies upon the lumbar spine. In the cadaver, if the small intestines be drawn firmly downward and backward so as to put the mesentery on the stretch, and then the gas be emptied from the stomach along the duodenum, its passage will be seen to be obstructed at this point, and the duodenum will become distended. Many conditions contribute to render this anatomical arrangement pathological. A condition of enteroptosis is usually present, so that the major part of the small intestines hangs in the pelvis, and this condition is more complete when the intestines are contracted and empty and when the abdominal wall is relaxed. Kundrat states that some peculiarity of the mesentery is necessary, and obstruction is more likely to occur when that structure is rotated, so that its sharp right margin is applied to the duodenum transversum. A low position of the duodenum appears to expose it more to pressure by the mesentery. Several cases have been the subjects of scoliosis, which rendered the spine more prominent, and the continued dorsal position appears to have contributed to the post-operative cases. In some of the cases of acute paralytic dilatation of the stomach, it would appear that the small intestines have been driven downwards and backwards into the pelvis by the enlarging organ, and the mesentery has been drawn tightly across the duodenum. In such cases, in addition to the stomach, the duodenum is distended to the point at which it is crossed by the mesentery, and a vicious circle is thus established. This cannot be accepted as the primary cause of all cases of acute dilatation of the stomach, nor does it always follow as a secondary result, for the dilatation frequently extends a variable distance down the jejunum, and profuse diarrhœa is often present in acute gastric dilatation (Campbell Thomson).

Various degrees of this condition are found. One of the pathologists at the London Hospital informs me that it is not uncommon to find dilatation and hypertrophy of the duodenum down to the point where it is crossed by the mesentery, in cases where the stomach is not much distended and no symptoms of the condition have arisen during life. A recent case (1906) exhibited this condition very well, and is recorded in the post-mortem reports (No. 1,311).

In the case of a woman aged fifty-nine (reported by Dr. W. Ewart) who died of uncontrollable vomiting and broncho-pneumonia, the small intestine, which was empty and contracted, occupied the pelvis. The duodenum was distended with thick, bile-stained contents as low as its transverse portion. The stomach had, however, been emptied by the vomiting, and was contracted at the autopsy. In fully developed cases the stomach is greatly distended, and may even reach to the level of the symphysis pubis.

Symptoms.—Many of the cases have followed abdominal operation, where we may suppose that the condition has been produced by a previous condition of enteroptosis, rendered active by paresis of the stomach due to the anæsthetic, the empty condition of the small intestine, and the maintenance of the dorsal position for a long time.

The onset of acute symptoms in other cases has been attributed to vomiting, straining, or even laughing.

The feature of these cases is the gastric dilatation, which as a rule produces an epigastric resonant swelling, which may be definitely identified as the stomach by appropriate means. The pulse as a rule is rapid and feeble, and the patient exceedingly weak.

Snitzler recognized and cured his case.

Treatment.—The position of the patient should be so arranged as to relieve the tension on the mesentery. The genu-pectoral position has been recommended, but the patient is seldom strong enough to assume it. In that event he should lie on his face or right side, whilst his pelvis is elevated by a pillow and the foot of the bed is raised. The lower abdomen may be manipulated to induce the intestines to pass upwards. The stomach should be emptied by a tube and washed out, and should distension recur, this may be repeated once or twice. Should these means fail, laparotomy may be performed, and various procedures, such as manipulations, gastrotomy, and gastro-enterostomy, may be carried out, but these are desperate measures.

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(iii.) **Internal Herniaform Incarceration of the Intestine.**—This group of cases of intestinal obstruction includes—(1) Obstruction due to bands, including Meckel's diverticulum; (2) obstruction due to apertures; (3) obstruction due to internal hernia. In all of these a similar part of the intestine is surrounded in the same way by a ringlike constriction. The symptoms are therefore almost identical, and the treatment is on similar lines. They are for this reason appropriately discussed together.

(A) **Intestinal Obstruction due to Bands.**—*The varieties of bands* are as follows:

1. Solitary peritoneal bands.
2. Omental bands.
3. Meckel's diverticulum and other diverticula of the intestine.
4. Normal structures abnormally attached, such as appendix vermiformis, appendicæ epiploicæ, Fallopian tube, hydatid of Morgagni, or the pedicle of an ovarian tumour or a fibroid.

Incidence.—The London Hospital records for thirteen years show, out of 669 cases of intestinal obstruction, 67 were due to bands other than Meckel's diverticulum. Of these 67, 34 were men and 33 were women; 36 cases died, giving a mortality of 53·7 per cent.

1. *Solitary Peritoneal Bands.*—These bands are formed by the moulding of the plastic lymph which is thrown out during an attack of peritonitis. They are usually the result of localized peritonitis. *The method* by which these localized attacks of peritonitis lead to the formation of a peritoneal band is illustrated by Fig. 16, in which is represented a coil of gut just reduced from a strangulated hernia, and lying paralyzed and inflamed against the abdominal wall adjacent to the ring. For two or three days this coil of gut will rest there inert, and will become adherent by plastic lymph to the parietal peritoneum. As it recovers its activity and contractility, it will be withdrawn once more to a higher position in the abdomen, drawing out the plastic exudate, and moulding it into a rounded cord as the coil of gut retreats from its former point of attachment. It is fortunate that in the vast majority of cases such cords are absorbed even when they have become organized into fibrous tissue; but in the minority these cords persist, and in some of these coils of gut are snared by them. The question of what induces lymph and adhesions to persist, whilst in the vast majority of cases it is

absorbed, is a very interesting one. Thus an operation during or soon after an acute attack of appendicitis, salpingitis, or peritonitis, shows that a large part of the peritoneum is an inextricable mass of adhesions; but when the acute attack has subsided, and a month or two has passed, another operation will show that nearly all these adhesions have been absorbed, and only a few persist around the source of irritation. In other cases dense adhesions persist, and may even become progressive, so as to invade the whole peritoneum, as in sclerosing peritonitis. The cause of the persistence of adhesions is persistence of the cause. In all other parts of the body chronic induration is the result of a persistent cause, and so it is in the peritoneum. Foreign bodies, whether in the appendix or the gall-bladder, maintain the inflammation and the adhesions. Pockets of pus which have no escape do so in the Fallopian tubes, whilst many cases of progressive adhesive peritonitis are tubercular in character, and contain their irritant, the tubercle bacillus, buried in their substance. Where the causal irritant ceases to exist, the adhesions will not long persist.

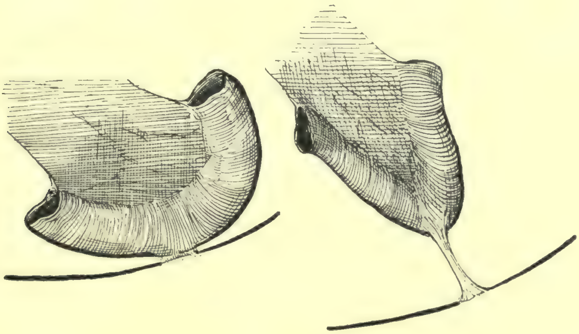


FIG. 16.—TO ILLUSTRATE THE FORMATION OF A PERITONEAL BAND.

The *situation* of such bands will be determined by the common sites of localized peritonitis. Many are formed in the region of the appendix, and not a few originate from inflammation of the uterus, the Fallopian tubes, or the ovaries. The hernial rings are the site of some, and the sigmoid of others, but these are often appendicæ epiploicæ attached at their extremities. Ulcers of the lower ileum, especially when tubercular, frequently give rise to patches of peritonitis on their obverse surface, and from these bands may spring, whilst tubercular glands of the mesentery, especially in its lower part, when they soften may similarly produce them. Bands are not seldom seen attached to the site of surgical procedures. General peritonitis, whether it originates from an appendix, a gastric ulcer, a gall-bladder, or especially when it is tubercular in origin, may leave behind a solitary band among many short adhesions. Malignant disease may lead to such an adhesion, as in a case I shall have to record.

The *shape*, *size*, and *length*, of such bands varies within the widest limits. They have been recorded a foot or more in length, and, on the other hand, may be no more than a fraction of an inch. Some are as thick as a finger, and some as thin as twine. Some are round, and others thick or filmy ribbons.

For the most part bands are attached at both ends, but they may break either at one end or at some part of their length, and then may reattach themselves, as is not infrequently seen, with Meckel's diverticula, or they may remain free at one end. In the latter case noosing or lassoing of a coil of gut is not likely to occur, unless the band terminates in a knob or excrescence of fat or fibrous tissue—a not unusual thing in the case of diverticula or elongated appendicæ epiploicæ.

Methods of Strangulation by Bands.—When the band is attached at both ends, a coil of gut may be incarcerated by it in three ways:

1. It may pass under it, and be constricted between the band and what is usually the posterior abdominal wall.

2. The gut may hang over the band, acting as a sling, as wet clothes are slung over a clothes-line.

3. Or, as is most common, the band may snare, knot, or noose, the coil of gut in any of the many forms of knot which may be formed from a piece of string attached at both ends.

4. In rare cases the band acts by *kinking* a coil of intestine to which it is attached, but this is usually considered as another variety of obstruction. It is not uncommon, however, to find that the obstruction is composite in a case of strangulation by band. In addition to encircling some other coil of gut, the band often kinks the coil from which it arises, and it is not rare for the strangulated coil to twist on its narrowed neck and form a volvulus.

The coil is probably seldom strangled when first it enters the snare, and must frequently escape before it is finally entrapped, but many circumstances tend to tighten the cord when once it is fairly on the coil. The band when once over the coil will naturally tend to slip up to the neck of it, and here it will compress the veins, and the coil will swell and become engorged and so further tighten the cord. Whatever makes the coil swell will draw further gut into the noose, and the mesentery to this gut will fill the constricting ring tighter and tighter, and increase the strangulation. This distension of the coil is no doubt first of all contributed to by contents from above being swept into it by peristaltic waves, and not so easily escaping from it. As the noose becomes tighter, however, the distension of the coil is chiefly due to the profuse and often blood-stained secretion sweated from its congested mucous membrane, and to the gas which is produced in its lumen. This gas is probably the result of uncontrolled bacterial fermentation, but is possibly derived from the asphyxial blood in the vessels, or is even the carbonic acid gas produced by the life processes of the tissues, which is not being removed by the obstructed blood-stream. In whatever way the distension is produced, it soon becomes as great as the tissues will permit, and chokes the ring formed by the band with the extra œdematous mesentery and gut it drags into it.

Although peritoneal bands are more often single, they are not rarely multiple, and the surgeon should not be content unless he can see that the constriction was at a point where dilated joins collapsed gut. It has not infrequently happened that the wrong band was divided, and another or even several were found at an autopsy. This deplorable error will always be avoided if collapsed gut from the cæcal region is drawn out and traced up to the point obstructed, and the unreliable method of following dilated gut downward as far as possible avoided.

Strangulation by band unfortunately complicates other acute abdominal conditions. A case will be narrated later in which it occurred during convalescence from acute tubercular peritonitis, and another four days after obstruction by cancer at the splenic flexure had been relieved by colotomy in the transverse colon. Acute obstruction may complicate appendicitis at any stage, and this obstruction is not infrequently due to bands.

I have only encountered three cases of solitary peritoneal band; indeed, of all forms of intestinal obstruction, it is the rarest in my experience.

CASE I.—Tubercular peritonitis complicated by acute strangulation by band.

This case has many points of interest. In the first place the little lad was thought to have obstruction, but was found at the operation to be suffering from tubercular peritonitis. His relapse about a week later was due to strangulation by band. The case also illustrates the danger of tubercular mesenteric glands.

Joseph S., aged five, was admitted to the London Hospital in August, 1902, as a case of intestinal obstruction. He had not previously been ill. The attack commenced fairly suddenly four days before with pain in the abdomen and vomiting, which had persisted since. His bowels were, however, open on the following day—that is to say, three days before admission. His temperature was 99° F., his pulse-rate 124, and his eyes ringed, but he did not appear collapsed and was in no pain. His tongue was clean. His abdomen was somewhat distended, and separate coils of gut could be seen. There was a considerable quantity of free fluid in the flanks and above the pubes. The rectal examination revealed nothing, and an enema gave no result. Before operation the diagnosis lay between intestinal obstruction and tubercular peritonitis.

Operation.—The abdomen was opened by a low median laparotomy, and much turbid fluid was evacuated. The peritoneum was studded with minute shotty tubercles more easily felt than seen. The abdomen was closed with through and through silkworm gut.

Progress.—The lad was at first very ill, but in a day or two, when his bowels had been well opened, he improved, and was convalescing, when, nine days after operation, he was seized with severe abdominal pain and vomiting. Three turpentine enemata gave no result. The symptoms were so like his previous attack that no diagnosis of intestinal obstruction was made for twenty-four hours, and then he was moribund. He died in thirty-six hours.

Autopsy.—A band was found stretching from the sigmoid to a mass of caseating mesenteric glands in the lower part of the mesentery, and about 6 inches of ileum were tightly strangled in a noose. The caseating mass of glands had apparently only recently ruptured into the peritoneal cavity, producing the acute attack of tubercular peritonitis.

CASE II.—Acute intestinal obstruction due to band. Operation without anæsthetic; recovery.

Ada D., a Jewess aged fifty-five, was admitted collapsed to the London Hospital in May, 1903. She did not speak English well, but apparently she had suffered from parametritis in Russia nine years before. An abdominal incision had been made above the left groin, which did not pass through the peritoneum. Her present illness had only lasted twenty-four hours. She was seized with violent abdominal pains, and had vomited continuously ever since, and the vomit was now becoming fæcal. Her bowels had not been open for twenty-four hours, and she had passed no flatus. Moreover, a turpentine enema gave no result. Her abdomen was not tender or distended, and a rectal and vaginal examination revealed nothing. Her general condition was very bad. Her pulse varied between 120 and 140, and at times could not be felt at the wrist. She was so collapsed that I determined to operate without anæsthesia.

Operation.—The skin incision was made with one sweep of a sharp knife. When the peritoneum was opened, dilated and collapsed coils of gut were found. The collapsed gut was rapidly traced up until it passed within a band, or, more correctly speaking, through the loop formed by a band uniting two adjacent coils of gut. This band was not more than $\frac{1}{4}$ inch long. It broke when forceps were placed on it. The ends were ligatured, but not invaginated, and the abdomen rapidly closed. The coil strangled in this case was undoubtedly the one which passed through the loop, but there also appeared to me to be kinking at the site of attachment of the band.

Strychnine (gr. $\frac{1}{7}$) was administered during the anæsthetic, and directly after the patient's return to bed 6 pints of normal saline solution were given by continuous subcutaneous transfusion.

Progress.—Her bowels were open the night of the operation, and by the next day her pulse was 88 and her condition extremely good. She made an uninterrupted recovery. My notes made at the time end with this remark : " A case practically saved by subcutaneous transfusion."

A curious thing about this case was that she remembered nothing of the operation, and, when questioned as to pain, said of course she had chloroform. A very little ether on an open mask was all the anæsthetic administered. She grumbled greatly about the removal of the stitches, which she said hurt her far more than anything she experienced at the operation.

Case III.—A band following appendicitis which produced kinking, volvulus, and strangulation over a band. Operation; recovery.

John J. M., aged nineteen, was admitted to the London Hospital in May, 1905. Two years previously, in February, 1903, he had suffered from an appendix abscess, which was incised and drained. The present illness had lasted four days. The onset was sudden, with acute pain chiefly referred to the umbilicus. He vomited soon after, and continuously until admission to the hospital, when the vomit was found to be fæcal. His bowels had been absolutely constipated, and a turpentine enema gave no result. His temperature was 98° F., his pulse 88, and his condition fair. His abdomen was distended, and more so in the right iliac region. The colon apparently took no part in the distension. An operation was immediately performed, and the abdomen opened in the middle line below the umbilicus. A most intricate arrangement of gut was found, for the elucidation and reduction of which it was necessary to bring out the lower 4 feet of the ileum. The arrangement was as follows: A band passed from the ileum, about 1 inch from the ileo-cæcal valve, to the under surface of the mesentery of a coil some 3 feet higher up. At its lower attachment it had kinked the ileum, whilst it had cut into and strictured the coil above where it crossed it. The distended coil above the band had passed down into the pelvis in front of it, and become incarcerated there, so that strangulation over a band was superadded. Furthermore, the three-foot loop of ileum between the points of attachment had turned over to the right and twisted the coil once in a loose volvulus. This coil was of a deep colour, but not as black or distended as a volvulus usually is. I divided the band, flattened out the mesentery, straightened the kinks, and twisted the volvulus. The patient made an uninterrupted recovery, and did not vomit once after the operation, which was doubtless due to the fact that his stomach was most carefully washed clean before chloroform was given.

This case well illustrates the complicated and cumulative character of some cases of strangulation by band. There were three degrees of distension of gut found in this case. The upper ileum was greatly distended and congested. The loop of ileum between the two attachments of the band was moderately distended, and the only contracted piece of small intestine was the last inch of the ileum between the kink and the ileo-cæcal valve.

The Great Omentum.—Of the functions of the great omentum very little has been written, and not much is definitely known.

Mr. W. McAdam Eccles has contributed a most valuable paper on this matter to the St. Bartholomew's Hospital Reports, vol. xxx. The great omentum is found in all mammalia and in many of the lower vertebrates. It grows faster than the other abdominal contents during life, and it is stated that before the age of forty-five years, in man, its lower border is on an average above the level of the pubic spine; after that age it is below it. The left border is lower than the right, and is therefore more frequently found in herniæ on that side. It lies behind that part of the abdominal wall which is unprotected by bone or thick masses of muscular tissue. Its lower border is fimbriated.

Its main function is probably a mechanical one, to permit of easy alimentary peristalsis and respiratory movements. It performs a similar function to the ligamenta alaria in the knee-joint. It is often overlooked that, if rounded objects like the intestines move over one another or change their shape and calibre, spaces must tend to be produced, and for these movements to be easy and facile these spaces must be readily filled by neighbouring organs, fat, or fluid. If none of these are available the movements are greatly hindered, for they are not powerful enough to produce a vacuum between the moving bodies. In the case of the brain the cerebro-spinal fluid fulfils this function of readily filling spaces of changing capacity. In the case of the knee-joint, as the spaces between the surfaces of the femur and the tibia vary with the different curvatures of the former in different positions, these spaces are readily filled by the ligamenta alaria and the semi-fluid pad of fat below the ligamentum patella, supplemented by a small quantity of synovial fluid which is normally present in the joint. In the abdominal cavity, however, fluid is not a practical solution to the problem, for in the erect position it would all gravitate into the pelvis, and would not be available unless in vast quantities. Under these circumstances a slippery membrane loaded with nodules of fat to give it the necessary bulk would appear the only solution to the mechanical problem of easy peristaltic movements in a closed cavity like the abdomen. As a peristaltic movement develops, and a triangular gap tends to be produced between that coil and the next, the omentum is sucked into the gap and piles up its fatty nodules until the gap is filled, and the movement progresses without any hindering suction on the outer surface of the gut. The converse is interesting when a dry peritonitis has made the omentum sticky and adherent, so that it is incapable of slipping, and no fluid has been poured out to, among other things, fulfil this function temporarily, the most absolute constipation results ; yet at the post-mortem we find signs that the intestines had struggled to contract in the lines of congestion and engorgement which mark the site of the potential gap between one coil and the next. In the absence of a sliding omentum or free fluid, the efforts of the gut to contract has sucked blood from its own wall into the space it attempted to create in order to complete its movement. A large part of intestinal obstruction in dry forms of peritonitis is probably due to stickiness and fixation of the great omentum.

Few organs in the body perform but one function, for Nature is far too economical of space and weight.

The second function of the omentum in order of importance is probably that of absorption, and perhaps secretion. The richness of the blood and lymph supply of the omentum support this view.

It may be that there is an active secretion of serum into the peritoneum, which is as actively sucked dry by the fimbriated margins of the omentum, and any bacterial or other infections rapidly dealt with by the lymphoid tissue between its layers and along the great gastric curve. It is at least certain that, when the peritoneum is infected by neoplasms or bacteria, the secondary deposits cluster on the omentum in a disproportionately large quantity.

It has been pointed out that when the stomach and transverse colon are distended the omentum is much shorter, for these organs pass between its layers and separate them. Along the great gastric curve the vessels and lymphatics are placed some inch or more from the curve in order to allow room for this expansion. The omentum may then be said to provide a reserve of peritoneum for the stomach and colon.

Under certain circumstances the omentum seems to be specially selected as a storehouse for fat.

Other normal functions have been suggested for it, which appear, however, rather fanciful. Such are—

To protect the intestines from violence and to keep them warm. It has even been ingeniously suggested that, when the gastro-epiploic vessels are flushed with blood during gastric digestion, the omentum becomes engorged, and warms up the small intestine as a preliminary to their stage of activity. The functions of the omentum, so far considerable, are normal ones which must be served each day; but there is a function which is seldom needed, but which is so beautifully served that the omentum must be specially adapted to fulfil it. This function is to localize infections of the peritoneum. For this reason the omentum has been called the "sticking-plaster" of the peritoneum. When an appendix is inflamed, the omentum often not only becomes adherent to it, but so enswathes it that it appears impossible that this can have been accomplished without the power of motility. The fimbriated extremities of the omentum appear to be provided for the purpose of searching out and plastering up all patches of peritonitis. Thus omentum is found adherent to any of the pelvic organs in the female which become inflamed (*vide* Fig. 17), to tubercular mesenteric glands about to perforate (*vide* Fig. 18), to perforating typhoid and tubercular ulcers, and to wounds of the gut, whether operative or accidental. This function is probably connected with the great absorbent powers of the omentum and its capacity to deal with bacterial infection. The whisp of omentum which plasters up a perforated appendix not only mechanically prevents the spread to pus, but, by a kind of exhaust action, drains away the serum and destroys the bacteria in it. This view is rather supported by the curious exemption

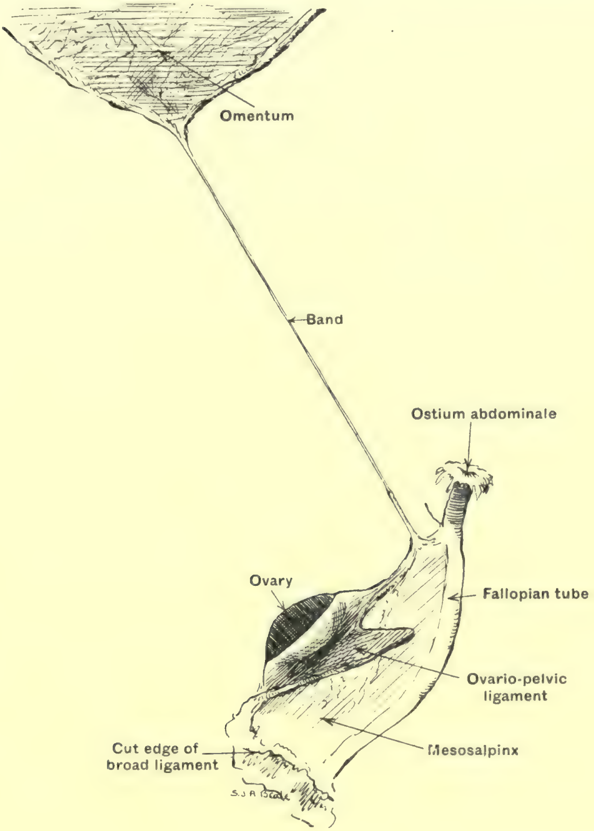


FIG. 17.—TO ILLUSTRATE AN OMENTAL BAND FORMED BY ADHESION OF OMENTUM TO AN INFLAMED FALLOPIAN TUBE.

from suppuration of omentum which has been herniated from a wound, and which has been replaced.

The great omentum also serves at times to prevent the protrusion of intestines from stab wounds. When included in a hernia sac with gut, it serves to protect the latter and mitigate the strangulation. Finally, it is a common channel for a collateral circulation when the portal radicles are obstructed by cirrhosis of the liver, doubtless owing to the fact that it so readily forms adhesions to parts which are connected with the systemic venous system.

2. *Omental Bands.*—Cases of bands derived from omentum are more numerous than those of isolated peritoneal adhesions.

An omental band differs from a peritoneal band in that one end has a constant attachment—namely, to the transverse colon or the general mass of the omentum. The attachment of the other end is to some patch of localized peritonitis, and these need not be reviewed again (*vide* Figs. 17 and 18).

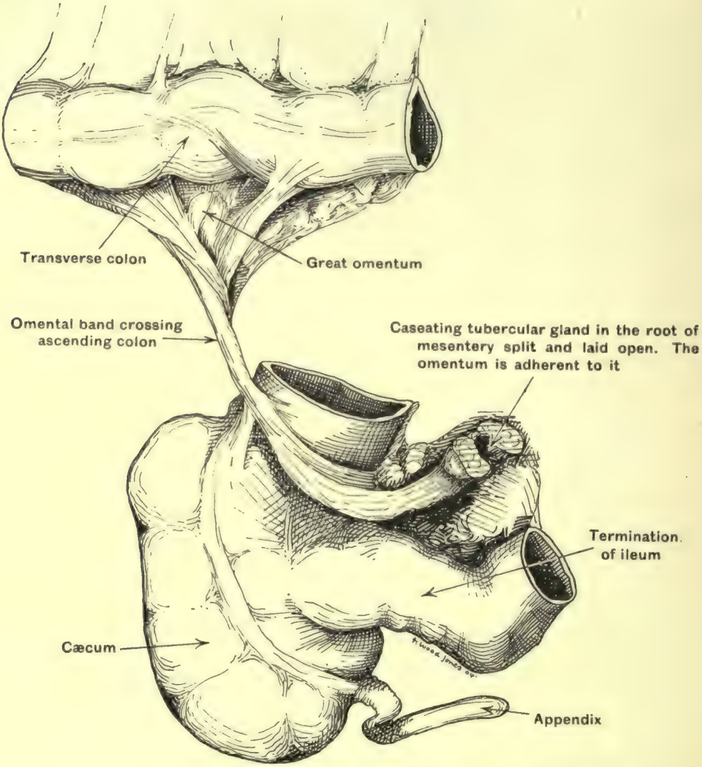

Transverse colon
Great omentum
Omental band crossing ascending colon
Caseating tubercular gland in the root of mesentery split and laid open. The omentum is adherent to it
Termination of ileum
Cæcum
Appendix
F. Wood Jones del.

FIG. 18.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM, SHOWING THE GREAT OMENTUM ADHERENT TO A TUBERCULOUS GLAND IN THE MESENTERY.

The adhesions between omentum and inflamed organs are absorbed and removed in a similar way to other peritoneal adhesions when the cause of inflammation has ceased. There is, however, this difference between peritoneal and omental bands: any short and close adhesion may lead to the formation of a band from the omentum passing to it, but peritoneal bands are only that small minority of peritoneal adhesions which are drawn out into cordlike structures. Moreover, a peritoneal band is an inflammatory structure, and liable to be absorbed. An omental band is a part of a normal structure, and not liable to be absorbed. This is probably another reason why omental bands are more common.

Omental bands are more often round, multiple, and arranged in a fan-shape, than peritoneal bands, and they are never very short. Strangulation by them does not seem to be so unyielding and cruel as when the band is attached at each end to gut or to such firm structures as abdominal wall or mesentery.

Omental bands are seldom drawn so tight round gut, apparently because of the mobility of the omentum and transverse colon, which permits that end of the band a considerable margin of slack.

The multiple character of omental bands should never be forgotten when dividing them, for one or more may easily be left. In Case III. several omental adhesions were divided in searching for a cause of the obstruction, but one was left which strangled a coil of ileum four days later, and led to the death of the patient. In a subsequent case, taught by the above sad experience, when I encountered multiple omental adhesions as a cause of obstruction, I enlarged the incision until the whole of the lower end of the omentum could be certainly identified, freed all its adhesions, and then rolled up the omentum and reduced it into the upper part of the abdomen. In dealing with omental adhesions, it should always be remembered that though cordlike thickenings occur in its substance, arranged roughly in a fan-shape, and doubtless along the lines of tension, yet the intervals between these bands is filled in by the thin film of the omentum, which effectively prevents gut passing through, and indeed, for mechanical purposes, makes the band a mere part of a flat membrane, and not a band at all. If the surgeon is misguided enough to divide the central part only of such an arrangement, he creates a rough aperture flanked on either side by definite bands. The rule, then, should be, If omental adhesions are divided, divide them all.

In the case of omental bands the gut may be strangled (1) under a band, (2) over a band; or (3) snared or noosed by a band; or (4) may pass between two bands through an aperture.

I have had four cases of intestinal obstruction due to omental bands, and all presented points of special interest. Of these four, three recovered.

CASE I.—A small boy, James T., aged twelve, was seen at the London Hospital in June, 1903.

The cause of the localized peritonitis was perfectly clear, for he had been in the London Hospital with appendicitis in November, 1900, and there was a history of a previous attack. He had now been seized with attacks of paroxysmal pain in the abdomen, and he vomited during the attacks. The pain was not in the right iliac fossa, but was chiefly umbilical in position. These attacks had lasted for three weeks, and for a fortnight he had been in bed. The bowels were said to be regular.

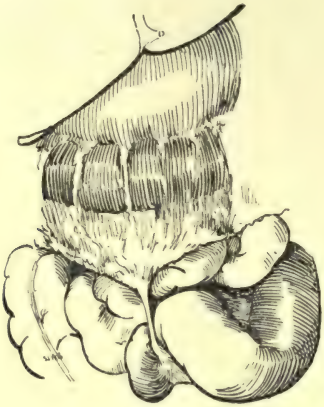


FIG. 19.—TO ILLUSTRATE STRANGULATION UNDER AN OMENTAL BAND ATTACHED TO THE MESENTERY (CASE I.).

On admission to the medical wards, where he remained a week before I saw him, his temperature was a little raised, but his pulse was normal. Beyond a little distension, no abnormality was made out in the abdomen. During this week he became steadily worse. The abdomen became greatly distended, and huge coils of intestine could be seen slowly peristalting. The bowels were slightly open twice daily with enemata, but the result consisted chiefly of mucus. The attacks of paroxysmal pain occurred about three times a day, and lasted about five minutes each time. He vomited irregularly with the pain. His pulse increased in frequency to 120 during this week, and his temperature during the last three days of it had steadily risen to 102°. On rectal examination it was found that the recto-vesical pouch was distended, but soft and not tender.

When I was asked to see him at the end of the week, it was clear he was suffering from a partial and chronic obstruction of the bowel, following appendicitis, and that this obstruction had lasted a month already. One mistake was made in diagnosis. Both from the size of the distended coils and from the extent of the distension, it was inferred that the point obstructed was in the great gut, and probably in the sigmoid, whereas it was in the lower ileum.

Operation.—As soon as the abdomen was opened, large coils of small gut, as large as normal colon, escaped from the wound. The cæcum was found collapsed. After tracing down a distended coil of gut for some 3 feet, it hitched, and when it came again showed the line of a band crossing it, and the junction at this point of dilated and collapsed gut.

The index-finger inserted along the upper surface of the mesentery encountered the band, which passed from the omentum to the under-surface of the mesentery (*vide* Fig. 20). The band was slack, and the intestine was constricted by it only on one side. This was clearly an example of strangulation beneath or over a band.

The cord was divided as high up as possible after a ligature had been placed on it and excised. The boy made an uninterrupted recovery. His bowels were open on the following day by enema, and after that naturally. His temperature fell to normal in four days, and he did not vomit at all after the anæsthetic.

CASE II.—Partial intestinal obstruction due to multiple omental bands, all of which were separated, and the omentum rolled up and reduced into the upper abdomen.

Mathilda P., aged thirty-eight, was admitted to the London Hospital in March, 1904. She had suffered from appendicitis four years previously, and again two years before, when an abscess developed, which was opened by Mr. Roxburgh by a middle-line incision.

Three days before admission she was seized with severe pains in the abdomen, chest, and back, and had vomited continuously ever since. When I saw her she was alcoholic, bronchitic, and apparently suffering in addition from a relapse of appendicitis and intestinal obstruction.

She was kept under observation for thirty-six hours, and then, when the vomiting continued to be incessant, enemata brought away neither fæces nor flatus, I felt compelled to operate. The incision was to the right of the old scar. The omentum was not only adherent to the abdominal wall, but to the whole of the cæcal and appendix region (*vide* Fig. 20). Apparently one coil of ileum, which was deeply congested and distended, passed between two omental adhesions, but the gut below this coil was not definitely collapsed, and that above not definitely distended. In such an inextricable muddle of small intestine and omental adhesion it was impossible to settle which was really the offending structure. It was then

I determined to free them all. I first tore the adhesions from the abdominal wall, and then, reaching across to the left edge of the omentum, drew the whole of the upper part of this structure out of the wound. I was then able to clearly see its attachments below and to separate them. There was some hæmorrhage, as the whole neighbourhood was inflamed.

The patient's condition did not permit the removal of the appendix. I closed the abdomen rapidly with through and through silkworm-gut sutures.

She vomited for three days steadily until the bowels were well opened. Enemata were given daily for five days, after which diarrhœa supervened. Her temperature was irregular for a fortnight and her condition bad, but her bronchitis had to be taken into account. Ultimately she made a good recovery.

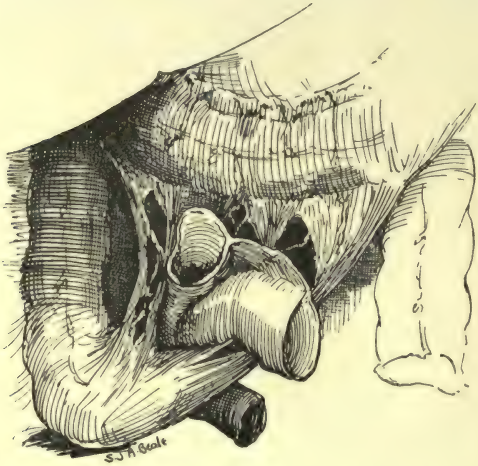


FIG. 20.—TO ILLUSTRATE STRANGULATION THROUGH AN APERTURE IN THE GREAT OMENTUM, THE RESULT OF INFLAMMATORY ADHESION (CASE II.).

CASE III.—Acute strangulation of the ileum by omental bands, complicating convalescence from a colotomy for carcinoma of the splenic flexure of the colon, probably due to the removal during the first operation of the central of several omental adhesions. Death.

Catharine P. was an old woman aged eighty-three, who was admitted to the London Hospital in April, 1902. For months she had suffered from increasing constipation, wasting, distension, flatulence, and colicky pains in the abdomen. During the previous thirty-six hours these symptoms had much increased; she had vomited frequently, and in spite of the use of enemata her bowels had not been open for three days. On examination her abdomen was found to be greatly distended, and the colon could be seen powerfully peristalting, especially the cæcum, which, when it contracted, formed a resonant tumour the size of a child's head. The small gut was apparently not distended. A turpentine enema brought away neither flatus nor fæces. A rectal and vaginal examination revealed nothing.

Operation.—A median laparotomy was performed at once. The small gut was very little distended. The cæcum was huge and tense; the transverse colon normal in size, but much hypertrophied; the sigmoid was collapsed and empty. It seemed clear that this was a case of acute dilatation of the cæcum, and a search was made for the obstruction in the region of the splenic flexure, where a mass was found in the upper part of the descending colon, adherent to the tissues around the left kidney. The middle of the transverse colon was drawn into the wound and a Paul's tube inserted.

During the examination of the cæcum old omental adhesions were encountered in the appendix region, and some two or three of these were divided between forceps; but I regret that no care was taken to divide them all, as they were obviously not the cause of the obstruction in the colon.

Progress.—During the next four days she did very well. At first the Paul's tube discharged a very large quantity of fluid fæcal matter. The vomiting ceased, and she appeared well on the way to recovery. On the night of the fourth day from the operation, she was seized with violent pain in the abdomen and became collapsed. The discharge of fæces from the Paul's tube ceased, she recommenced to vomit, and her temperature fell to subnormal. I did not see her until the following day, and then she was too feeble for operation. We thought that probably perforation had occurred from a stercoral ulcer either in the cæcum or just above the growth.

But a second and acute obstruction was discussed and thought to be very probable. She showed much more vitality than we had anticipated, and lived forty-eight hours after the onset of the acute obstruction.

The *Autopsy* was hurried and left much to be desired. Three feet of the lower ileum was found tightly strangled by an omental band, and gangrenous general peritonitis was present. The examination of the colon was apparently overlooked.

Case IV.—Strangulation of nearly the whole of the small intestine by an omental band, which passed like a sling beneath the mesentery from one end of the transverse colon to the other.

Eliza M., aged sixty-three, was admitted to the London Hospital in June, 1901. She had been wasting for six months, and had suffered from obscure abdominal discomfort. She gave no history of an injury to her abdomen. Three days before she was admitted she had been seized with severe paroxysmal pains, which lasted about five minutes, frequently recurred, and were associated with vomiting. She had passed neither fæces nor flatus since the onset of the pain, and an enema produced no result. Her abdomen was distended, and large coils of peristalting gut were seen through the emaciated abdominal wall.

The Operation.—The abdomen was opened by a median low incision. Red, œdematous and distended gut presented. The sigmoid, transverse colon, and cæcum, were empty and contracted. Contracted ileum was followed up from the cæcum for a distance of about 6 feet, when it hitched, but with a little traction came again and showed the impression of a slack band and the junction of distended and collapsed gut. A hand passed up the upper surface of the mesentery encountered no band, and after a couple of feet of distended gut had been drawn out with ease, and the contents began to gurgle past the constricted point, I was tempted to close the abdomen and regard the case as strangulation beneath a slack band, which could not readily be found. The second line of constriction on the gut had, however, not been seen, and only two of the three varieties of gut usually encountered in cases of obstruction. In other words, I did not know whether to regard the distended and congested intestine which filled the field of operation as gut within the obstruction or above it. I therefore prolonged the incision above the umbilicus and pulled down a coil of jejunum near its commencement. This coil was found to be much more distended, but less congested, than the gut below, and on following it down about 2 feet it hitched, and a hand passed up its mesentery encountered a band passing from the omentum in the region of the splenic flexure beneath nearly the whole of the mesentery, and up to join the omentum again about the hepatic flexure (*vide* Fig. 21). This band was divided, disentangled, and the ends ligatured and cut as short as possible. The band, which was some 6 inches or more in length, was rounded and very thin at its centre. It was extremely slack, and must have produced its effect chiefly by mildly constricting the veins of the large piece of mesentery which had insinuated itself over it, and, further, by kinking the gut at the lower of the two points where the band crossed the intestine. It is probable that it was of long duration and commenced as a spontaneous or traumatic perforation of the omentum. Through this a coil of gut insinuated itself, and was followed by more coils and their mesentery. The band became rounded and cordlike, and at last the contents so completely filled it that the vessels became constricted and subacute symptoms of obstruction supervened.

The patient did not vomit after the anæsthetic, and her bowels were open next day. She made a good recovery, complicated, however, by some bronchitis, which rendered it necessary to sit her up and give her strychnine in large doses.

This case should, perhaps, have been classed as strangulation through an aperture, but the hole was so large, and the obstruction so definitely due to the cordlike lower margin, that I have preferred to place it here.

3. *Meckel's Diverticulum and Other Vitello-Intestinal Vestiges: Development, Pathology, Symptoms, and Treatment, of its Numerous Varieties.*—Meckel's diverticulum is the persistent neck of the yolk-sac which passes with the vitelline vessels from the umbilicus to the ileum in an early stage of fœtal development. This structure should be obliterated in the human being during the sixth or seventh week of fœtal life. This obliteration is, however, often arrested at various stages, and a most wonderful variety of pathological curiosities are thus produced. Not all of these produce intestinal obstruction, but so many are associated with that condition that it seems advisable to describe here all the varieties of vitello-intestinal vestiges.

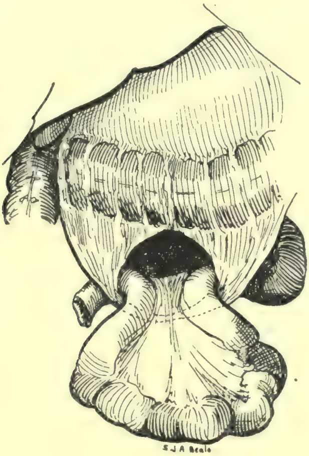


FIG. 21.—TO ILLUSTRATE THE CONDITION FOUND IN CASE IV.

The Development of the Yolk-Sac and Meckel's Diverticulum.—The following account is from the description by Professor Arthur Keith in his book on "Human Embryology and Morphology," and for the figures I am indebted to his kindness.

At the commencment of the third week the embryo appears to be situated like a cap upon the yolk-sac. At the end of the third week the alimentary canal shows division into three parts: (1) The fore-gut; (2) the hind-gut; (3) the mid-gut.

(1) The fore-gut is a short anterior diverticulum from which is developed later the pharynx, œsophagus, stomach, and that part of the duodenum which is anterior to the opening of the common bile-duct.

(2) The hind-gut is a short posterior diverticulum which later forms the rectum, sigmoid, and descending colon. Its end takes part in the formation of the cloaca, and from this the allantois grows out.

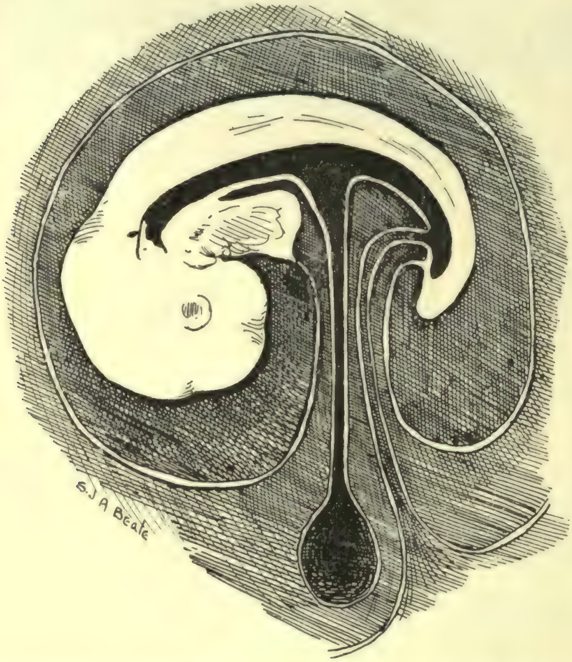


FIG. 22.—DIAGRAM TO SHOW THE DEVELOPMENT OF THE YOLK-SAC IN THE FIFTH WEEK OF FŒTAL LIFE.

(3) The mid-gut at the end of the third week forms the roof of the yolk-sac, and from it is formed all that part of the gut which lies between the opening of the common bile-duct in front and the splenic flexure of the colon behind.

The yolk-sac reaches its greatest size in the fourth week. The vitelline artery, the proximal part of which afterwards forms the superior mesenteric artery, ends on its walls. The vitelline veins commence on them.

Fig. 22 represents the stage of development which is reached in the fifth week of fœtal life. The mid-gut has been produced as a U-shaped loop from the roof of the yolk-sac. The neck of the yolk-sac has been drawn out into the long vitello-intestinal canal, and is attached to the apex of the U. This canal lies in the umbilical cord. If the neck of the yolk-sac persists, it forms Meckel's diverticulum. The yolk-sac itself, owing to the constriction of the umbilical orifice and the growth of the cord, comes to lie on the placenta.

By the sixth week the sac is in a state of retrogression. The U-shaped intestinal loop is at first extra-abdominal, and is situated within a funnel-shaped cavity formed by the somatopleure at the umbilicus.

The persistence of this stage is shown in Fig. 37, Case III., where it produced a congenital umbilical hernia and a congenital stricture of the gut above a Meckel's diverticulum.

The proximal part of the vitelline artery, which supplies the U-shaped loop, persists normally as the superior mesenteric artery, which, it will be remembered, supplies, in addition to its mesenteric branches, the ileo-colic, colica dextra, and colica media, to those parts of the colon derived from the mid-gut.

When a Meckel's diverticulum is present, it frequently has a mesentery which contains a further persistent part of the vitelline artery. This mesentery and artery, in crossing the ileum to join the superior mesenteric artery, twist, and sometimes stricture the gut (*vide* Fig. 23). Very rarely the neck of the yolk-sac may be absorbed, and yet the vitelline vessels may persist as a cord passing from the umbilicus, not to the free edge of the ileum, but to its mesentery. The fibrous remnant of the vitelline artery joins the superior mesenteric artery. This condition is illustrated in Fig. 38.

The Pathological Conditions which may result from Various Degrees of Persistence of the Yolk-Sac and its Vessels.—These are very numerous, and are best classified as follows :

(1) Diseases of the umbilicus due to persistence of the vitello-intestinal canal.
(2) A Meckel's diverticulum is present, and is attached to the umbilicus.
(3) A Meckel's diverticulum has broken free from the umbilicus, and is unattached at one end.
(4) The free end of a Meckel's diverticulum has formed a new attachment.

(5) A congenital stricture of the ileum exists in connection with a Meckel's diverticulum.

(6) The Meckel's diverticulum has entirely disappeared, but the vitello-intestinal vessels have persisted as a fibrous cord.

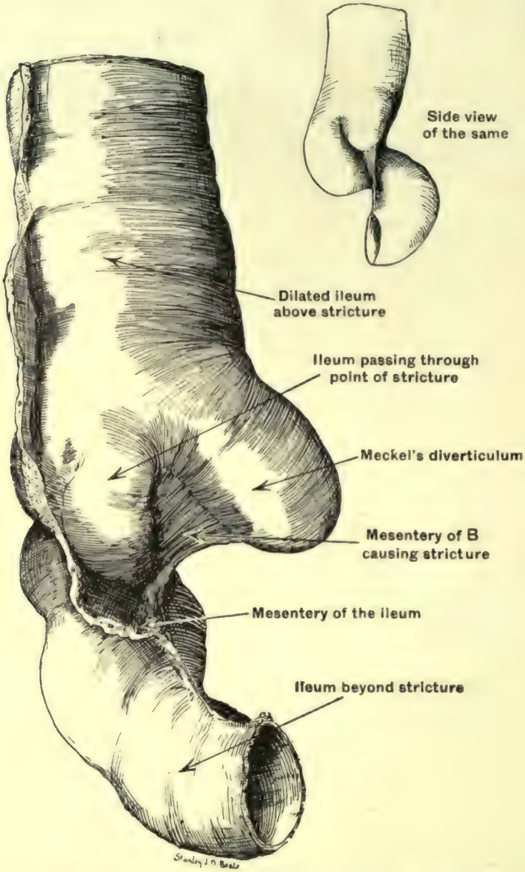


FIG. 23.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM, SHOWING THE ILEUM TWISTED AND STRICTURED BY A SHORT MESENTERY PASSING FROM MECKEL'S DIVERTICULUM TO THE MESENTERY OF THE ILEUM.

(1) *Diseases of the Umbilicus due to the Persistence of the Vitello-Intestinal Canal.*—(i.) *An adenoma or enteroteratoma* may be formed from a vestige of the canal left at the umbilicus (*vide* Fig. 24). They were first described by Kolaczek in 1875 (*Journal of the Pathological Institute of Breslau*), and afterwards by Küstner in 1877 (*Virchow's*

Archives). The tumours are first noticed about the third month after birth. They are the size of a raspberry, red, firm, elastic, and secrete an alkaline viscous fluid. For the most part they are pedunculated, and cannot be reduced into the abdomen.

Bergmann claims that they are short vitello-intestinal sinuses closed at their deeper end, and with the mucous membrane everted. He states that the base, when broadly pedunculated, may contain a process of peritoneum, and gut may occupy this.

When removing them it is necessary to bear this in mind, in order to avoid leaving the peritoneal cavity open or even wounding a coil of small gut in this sac. The red surface of the little tumour is studded with Lieberkühn's glands, which secrete the viscid fluid. A muscularis mucosa is found below this. The centre is formed of plain muscle fibre, which, according to some, is disposed in a longitudinal and circular layer. A dimple often exists at the apex, and this will sometimes admit the tip of a fine probe.

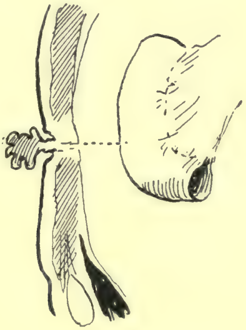


FIG. 24.—DIAGRAM TO ILLUSTRATE AN ADENOMA FORMED FROM A VESTIGE OF THE VITELLO-INTESTINAL CANAL LEFT AT THE UMBILICUS.

Adenomata must be distinguished from granulomata, which appear about the fifth week, are softer, do not secrete mucus, and are composed of granulation tissue.

(ii.) *Columnar-celled carcinoma* may probably develop even late in life from such a vestige. Such a case is described in the Pathological Society's Transactions, vol. l., p. 243, by Dr. C. D. Green. It was excised, and the patient recovered.

(iii.) *The vitello-intestinal canal may remain open at both ends, and a fæcal fistula result* (*vide* Fig. 25). The proportion of alimentary matter which escapes from the umbilicus varies very greatly. When a congenital stricture or occlusion exists at the ileo-cæcal valve, the whole of the fæces may be so discharged, and the condition is then known as *congenital umbilical anus* (Bergmann). More often only a part is so discharged, and when the orifice is small, only a small part, and this only at intervals. The discharge may commence some time after birth, apparently as the result of inflammation of the umbilicus, coughing, or straining.

When the escape of fæcal matter is small and the child is holding its own, treatment is better postponed, or limited to destroying the lining mucous membrane of the accessible part of the tube with actual cautery. Mr. Bernard Pitts reports such a case in the Transactions of the Pathological Society, vol. xxiii., p. 145, which appeared at the third week after birth, and closed spontaneously at the seventh year.

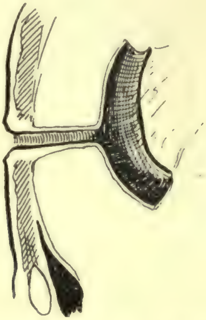


FIG. 25.—DIAGRAM TO ILLUSTRATE THE VITELLO-INTESTINAL CANAL OPEN AT BOTH ENDS.

When the discharge is excessive and persistent, and when the child's condition permits, the umbilicus and canal may be excised, or the orifice may be closed by a plastic operation. Vitello-intestinal fistulæ are readily distinguished from urachal and gastric fistulæ by the nature of the fluid which escapes.

(iv.) *Various degrees of prolapse* occur when the opening of the canal at the umbilicus is wide. The canal alone may prolapse so that there is a single orifice, or it may be so completely extroverted that the orifice of the upper and lower end of the ileum may appear on the tumour so produced, and these may proceed to even further prolapse. Fæcal matter is discharged from the upper orifice alone. Such a prolapse should be reduced and retained in position until the age and condition of the child permit of a resection of the umbilicus and the canal, or some less severe plastic operation.

(v.) *Intussusception* may occur in the coil of ileum above or below the open canal, and project from the umbilicus. When the intussusception is in the upper coil of ileum, intestinal obstruction will result; but when it is in the lower coil, as in the following case, the results will chiefly be marasmus from a small gut fistula. Mr. Golding-Bird has reported and figured such a case in the Transactions of the Clinical Society, 1896, p. 33. The intussusception projected from the lower coil of ileum of a prolapsed canal in a four-weeks-old child. A probe could be passed all round it between the prolapsed mucous membrane of the canal and the intussusception. Fæcal matter escaped freely from the upper coil of ileum by the side of the intussusception. Reduction was impossible, and the child died in three days.

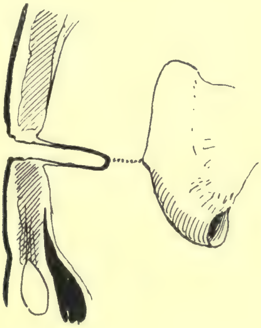


Fig. 26.—Diagram to illustrate the Vitello-Intestinal Canal closed at its deeper end.

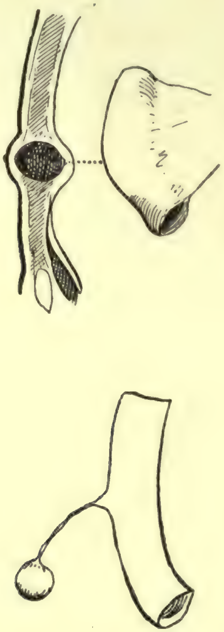
(vi.) *The canal may be closed at its deeper end*, and even separated from the gut, but remain open at its umbilical end, and discharge secretion from the Lieberkühn glands and solitary follicles which line it (*vide* Fig. 26). This secretion is viscous, mucoid, and alkaline, and serves to distinguish such a fistula from a urachal fistula, or the very rare gastric fistula which discharges an acid fluid which erodes the skin. This form of vitello-intestinal fistula may be treated by cautery or excision.

(vii.) In very rare cases a *cyst* is formed by the occlusion at both ends of a part of the canal and its subsequent distension. Such a cyst should be excised (*vide* Fig. 27). The diagnosis is made subsequently from a microscopic section of the wall and the character of the contents.

(2) *A Meckel's Diverticulum is present, and is attached to the*

Umbilicus.—This is an uncommon form of Meckel's diverticulum (*vide* Fig. 28). Cazin (" Études sur les Diverticles de l'Intestine," Paris, 1862) found this attachment in but three of twenty-three cases.

Among the thirteen cases and specimens upon which this account is based, in only two was the end attached to the umbilicus. The first was a fibrous cord due to persistence of the vitelline vessels only. In the second case the diverticle and the adjacent coils of gut were in a congenital umbilical hernia, to the apex of which the diverticle was attached. The diverticulum as a rule extends but a part of the way from the edge of the ileum to the umbilicus, and is then continued as a fibrous cord (*vide* Fig. 28).



FIGS. 27, 27*a*.—DIAGRAMS TO ILLUSTRATE CYSTS FORMED AS THE RESULT OF IMPERFECT OBLITERATION OF THE VITELLO-INTESTINAL CANAL.

As to the manner in which a diverticle so attached can lead to obstruction :

1. Incarceration as under a band would seem to have occurred in Fig. 38, where the band deeply constricted the ileum at the

point it crossed it. This specimen was obtained from the body of an ancient female in the dissecting-room, and no history was available, but the gut above the constriction did not show hypertrophy or distension. The tendency is to regard a Meckel's diverticulum which is attached to the umbilicus as horizontally disposed; but its direction is more nearly vertical, for the last 3 feet of ileum are normally situated in the pelvis.

2. Strangulation over such a diverticulum would appear impossible.

3. Noosing and knotting of coils of small gut above or below occurs in precisely the same fashion as with solitary peritoneal bands. The great length and looseness of the diverticular cord permit of very complicated knots.

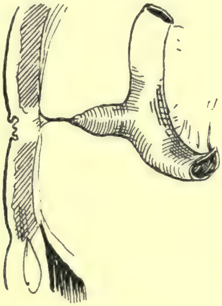


FIG. 28.—DIAGRAM TO ILLUSTRATE THE ATTACHMENT OF MECKEL'S DIVERTICULUM TO THE UMBILICUS.

4. When the condition persists in which the vitello-intestinal canal is still attached to the umbilicus, and occupies a funnel-shaped cavity in the root of the cord, antenatal strangulation may occur. The simultaneous growth of the intestine and contraction of the umbilical ring lead to stricture of the gut. A congenital strangulated umbilical Littré's hernia was thus produced.

(3) *A Meckel's Diverticulum has broken free from the Umbilicus, and is unattached at One End.*

(i.) Meckel's diverticulum is found in 2 *per cent.* of all human subjects, and in the great majority of cases it exists as a free thimble-like projection, producing no symptoms. This projection always springs from the *free border of the gut*, and diverticula found in other situations are not derived from the vitello-intestinal duct.

Its *position* is on an average 20 inches from the ileo-cæcal valve in the adult body. Professor E. von Bergmann gives the limits as 12 to 52 inches. Mr. Bilton Pollard has, however, described a case in the Transactions of the Pathological Society, vol. xlvii., p. 47, where the diverticle arose 24 inches below the pylorus, and terminated at the umbilicus after a course of 3 feet.

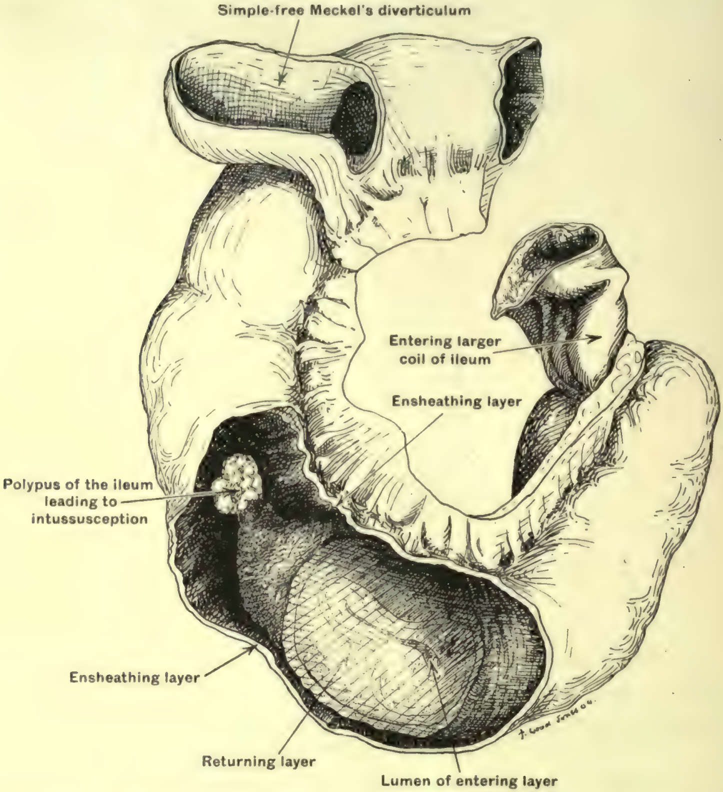

Simple-free Meckel's diverticulum
Entering larger coil of ileum
Ensheathing layer
Polypus of the ileum leading to intussusception
Ensheathing layer
Returning layer
Lumen of entering layer

FIG. 29.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM.

The *structure* of the wall of the diverticulum is identical with that of the lower end of the ileum. The peritoneal muscular layers and mucous membrane are similar. Solitary follicles, and even Peyer's patches, have been found in the latter. The opening into the ileum is sometimes valved, though as a rule it is the widest part of the diverticle, and as wide as the ileum at the point of attachment.

The *shape* of a free Meckel's diverticulum is most often that of a thimble, but in rare instances it has been cylindrical, pear-shaped, or its end has been enlarged by secondary diverticula so that it bore some resemblance to a hammer.

I have encountered two cases of carcinoma of the ileum some 4 feet from the ileo-cæcal valve, which produced intussusception and death. Curiously enough, in both cases a simple free Meckel's diverticulum was found below the intussusception. One of these specimens is shown in Fig. 29.

(ii.) A free diverticle when *it is of a pear shape* may become so distended as to interrupt the lumen of the ileum at its point of attachment, and thus produce intestinal obstruction.

(iii.) In other instances *volvulus* has been produced by such a distended Meckel's diverticulum rotating on its axis. I have once encountered volvulus of a Meckel's diverticulum, but in this case the distal end was attached to the mesentery by a short fibrous cord. A coil of gut had passed below the diverticle, and as more and more gut was drawn into the ring it had turned the central part of the diverticle over three times, and thus produced volvulus of both ends in opposite directions. The diverticle was gangrenous. As in ordinary cases of volvulus of intestine, the symptoms will be a combination of acute intestinal obstruction rapidly followed by general peritonitis from gangrene of the diverticulum.

(iv.) *The mesentery of a free Meckel's diverticulum may twist, kink, or stricture the ileum* as it passes across it to join the genital mesentery. Such a specimen is shown in Fig. 23. I owe this old specimen to the kindness of Professor Arthur Keith, who found it wrongly classified in the London Hospital Museum and dissected it.

I have operated on a similar case:

A Meckel's diverticulum the mesentery of which produced an acute kink of the ileum, found in a right inguinal hernia (Littré's hernia), and reduced. Death.

Stephen C., aged four months, was admitted to the Metropolitan Hospital in January, 1900. He had suffered from a right inguinal hernia since birth. Vomiting and total constipation had existed for two days, and the hernia was irreducible, tense, and there was no impulse in it. The child was said to be a premature birth at the sixth month. The father suffered from hernia, and six out of seventeen brothers and sisters.

Operation.—I opened the sac. The major part of the coil of gut in the sac was of a pinkish colour, and apparently not strangled by the neck of the sac. One part of the coil, which afterwards was found to be the diverticulum, was deep purple, and adherent to the wall of the sac. The mesentery was blood-stained and purple where the diverticulum joined it. I did not recognize the condition, freed and reduced the gut, and tied off the neck of the sac. The symptoms continued unabated, and the child died in four hours.

The specimen which is shown in Fig. 30 was obtained at the autopsy, and a careful dissection then showed that a Meckel's diverticulum was present, the mesentery of which had kinked the coil of ileum below it. The occlusion of the gut was not complete, however, until the whole mass became incarcerated in a right inguinal hernia. This is an example of Littré's hernia—that is to say, a hernia containing a Meckel's diverticulum.

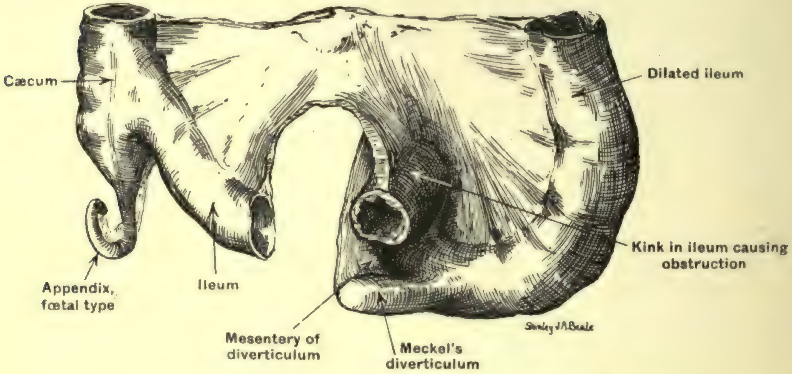


FIG. 30.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM.

The mesentery of the Meckel's diverticulum has produced a kink in the ileum below it.

(v.) A free Meckel's diverticulum may produce an *intussusception of the ileum* in the following manner: The diverticle may become inverted into the lumen of the ileum, in which it projects as a polypoid mass. It is then grasped by the gut below and drawn on down the alimentary canal, producing an invagination of the gut above. Professor Arthur Keith, late Curator to the London Hospital Museum, has investigated the intussusceptions of the ileum mounted in that museum, and found several unrecognized examples of this condition. One of these has been unravelled, and is shown in Fig. 31. It clearly shows a Meckel's diverticulum turned inside out into the ileum, and forming the apex of the intussusception.

(vi.) *A Meckel's diverticulum* may exist as a narrow tube or band, free at one end, and *terminating in a rounded knob or ampulla.* This band may one day encircle a coil of gut, and having become knotted, the swollen end will prevent this knot coming undone, in just the same way as a shopkeeper places a knot at the end of a string with which he is making up a parcel, to prevent its slipping.

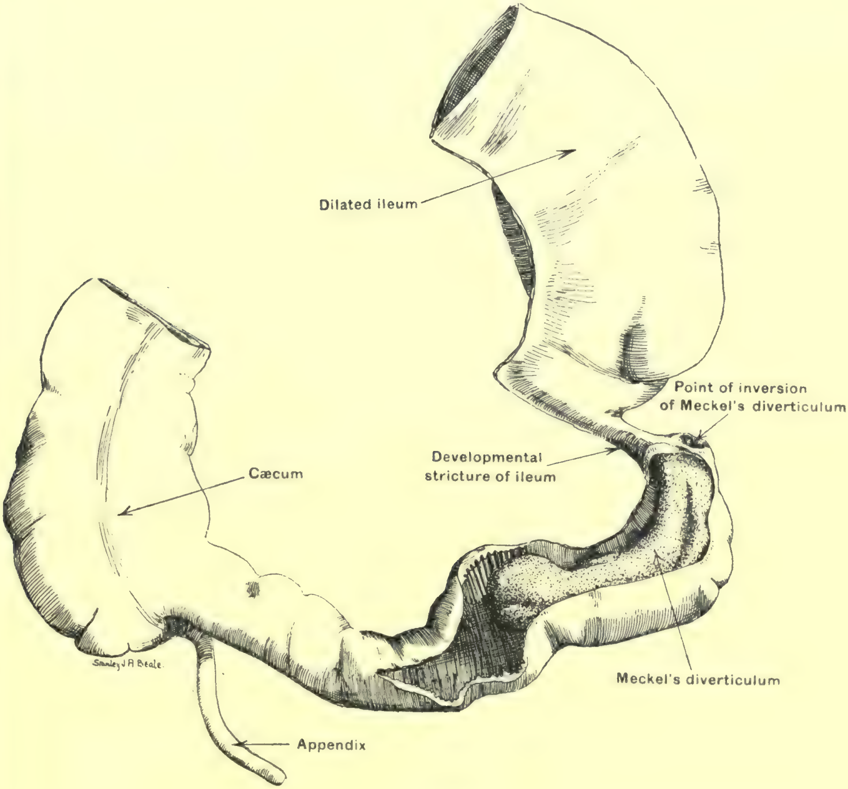

Dilated ileum
Point of inversion of Meckel's diverticulum
Developmental stricture of ileum
Cæcum
Stanley J A Beale
Meckel's diverticulum
Appendix

FIG. 31.—FROM A SPECIMEN IN THE MUSEUM OF THE LONDON HOSPITAL, ILLUSTRATING INVERSION OF MECKEL'S DIVERTICULUM, WHICH PRODUCED AN INTUSSUSCEPTION OF THE ILEUM, ALSO CONGENITAL STENOSIS OF THE ILEUM ABOVE THE DIVERTICULUM.

The intussusception has been reduced to show the inverted diverticulum.

This form of diverticle is very rare, but a good specimen exists in the museum of the London Hospital, which is represented in Fig. 32, and another specimen will be found in the Royal College of Surgeons Museum, No. 2695B.

The knots formed by such diverticula may be of several varieties and very complicated. They have been fully described by M. Parise (*Bull. de l'Acad. de Méd.*, tome xvi., p. 373).

(vii.) *A free Meckel's diverticulum may be found in a hernia sac* (*Littré's hernia*). I have encountered a diverticulum in a congenital inguinal and an umbilical hernia.

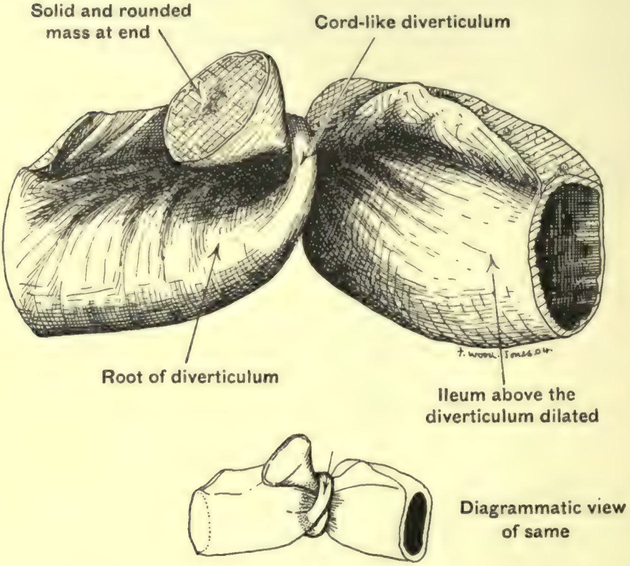

Solid and rounded mass at end
Cord-like diverticulum
Root of diverticulum
Ileum above the diverticulum dilated
Diagrammatic view of same

FIG. 32.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM.

The diverticulum may become strangulated, and lead to one form of partial enterocele.

(4) *The Free End of a Meckel's Diverticulum has formed a New Attachment.*—This is the form of Meckel's diverticulum which most commonly produces internal strangulation, and in the great majority of cases the attachment is to the mesentery of the small intestine (*vide* Fig. 33).

Point of Attachment.—Cazin collected twenty-three cases, and found the point of attachment as follows:

Near the umbilicus	3
Near the inguinal ring	1
To small gut	6
To cæcum	2
To colon	1
To mesentery	10

It will be seen that half the cases not attached to the umbilicus were attached to the mesentery. The attachments of my three cases of this variety were—

To the mesentery	2
To the omentum	1

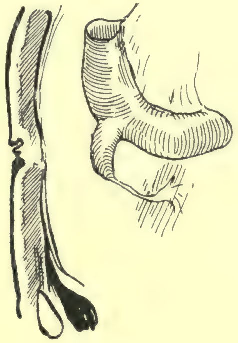


FIG. 33.—DIAGRAM TO ILLUSTRATE THE ATTACHMENT OF MECKEL'S DIVERTICULUM TO THE MESENTERY.

Attachment has also been recorded to the bladder, pelvic organs in the female, hernial rings, appendix region, and the abdominal wall other than the umbilicus. Localized peritonitis appears to have determined the point of attachment in many cases, and, just as in the formation of omental bands, glandular disease in the mesentery plays an important part.

The fresh attachment of the diverticle to the mesentery may be either above or below its root. The *length* of the diverticulum varies within wide limits.

Forms.—In form it presents three varieties. Most commonly it is patent for half its length, and terminates in a rounded cord which may possess a small lumen for a variable distance from the open diverticulum.

Less commonly the diverticulum is open to its point of attachment, and still less commonly the diverticulum is obliterated throughout its whole extent, and exists only as a fibrous cord.

This cord must be distinguished from that derived from the vitello-intestinal vessels alone by the attachment of its root to the free margin of the ileum, whereas the purely vascular cord passes over the ileum to become attached to the mesentery, where it is continuous with the superior mesenteric artery.

Methods of Strangulation—(i.) *Strangulation beneath a Band.*—It is generally agreed that this is the commonest form of obstruction encountered in this variety of Meckel's diverticulum. I have seen it twice in my three cases.

It is as a rule a mild variety. The obstruction is partial only, and the vessels are not greatly interfered with unless it be allowed to persist a long time.

(ii.) *Strangulation by Noosing and Snaring.*—The length which such a structure may attain enables it to form the most fantastic nooses in some instances. As a rule, however, the band is arranged in a simple spiral, but intricate descriptions have been given of loops so arranged that coils of gut passed through several apertures. A true figure-of-eight arrangement has been described.

(iii.) *The Meckel's diverticulum very frequently strangles itself, and becomes distended and gangrenous.* To realize this one must remember that its structure is like any other coil of small gut. It possesses a large lumen crowded with glands and swarming with micro-organisms. It has a good blood-supply. Moreover, the common form, half diverticle and half cord, lends itself readily to strangulation when it is twisted, so that the sharp cord is drawn across the base of the vascular diverticulum. Such a case is shown in Fig. 35, Case II. In another case a coil of gut, as it passed below the arcade of the diverticulum, rolled it over and over three times, producing a volvulus at either end, and gangrene of the diverticulum between (*vide* Fig. 36, Case III.).

The result of this strangulation and gangrene of the diverticulum itself is early and general peritonitis, which complicates the diagnosis.

(iv.) *Volvulus of the coil of gut incarcerated by the band* is not infrequent, and plays a part in the production of the intestinal obstruction.

(v.) *The ileum may be kinked or twisted at the point of origin of the diverticulum* in consequence of the powerful traction or twisting at this point.

The complexity and cumulative character of the obstruction from a Meckel's diverticulum will be seen to be an exact counterpart of that we have already discussed under Peritoneal Bands.

The Symptoms and Diagnosis of Obstruction due to a Reattached Meckel's Diverticulum.—In the majority of cases the patient will be a young male who has not previously suffered from localized peritonitis.

When the strangulation is beneath a diverticulum, the onset will be less sudden and severe, and the progress of the case less rapid. The bowels may still be partially opened, and the intestine above may have time to hypertrophy, distend, and appear as peristalting coils of gut, attended by colicky attacks of pain.

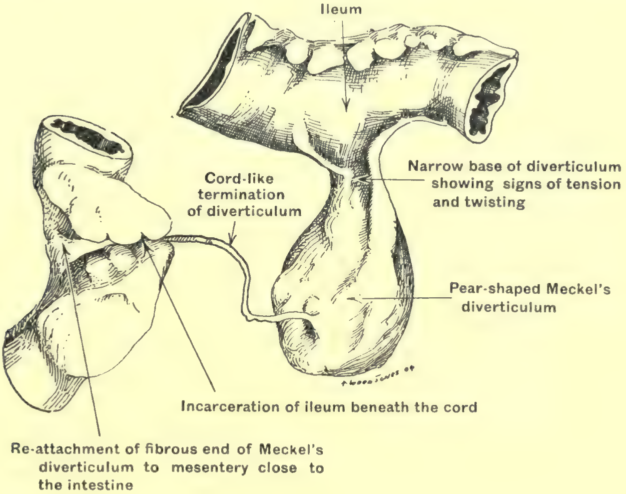

Ileum
Cord-like termination of diverticulum
Narrow base of diverticulum showing signs of tension and twisting
Pear-shaped Meckel's diverticulum
Incarceration of ileum beneath the cord
Re-attachment of fibrous end of Meckel's diverticulum to mesentery close to the intestine

FIG. 34.

When the diverticulum snares or nooses a coil of gut, the symptoms will be those of acute strangulation, as by any other form of band or orifice.

The tendency of the diverticulum itself to early gangrene will always be liable to superadd the symptoms of general peritonitis to those of acute obstruction. In such cases, in addition to profuse vomiting and total constipation, general rigidity and tenderness of the abdomen are present, some pyrexia, and the characteristic pulse of peritonitis.

Cases and Specimens.—Fig. 34 represents a specimen in the museum of the London Hospital. The Meckel's diverticulum springs from the free border of the ileum. It is of the somewhat unusual pear shape. Its constricted root shows signs of having been folded or twisted. From the end of the swollen ampulla a narrow band springs, and passes to be attached to the mesentery of an adjacent coil of ileum. It will be seen that this cord crosses and constricts the coil of ileum to whose mesentery it is attached.

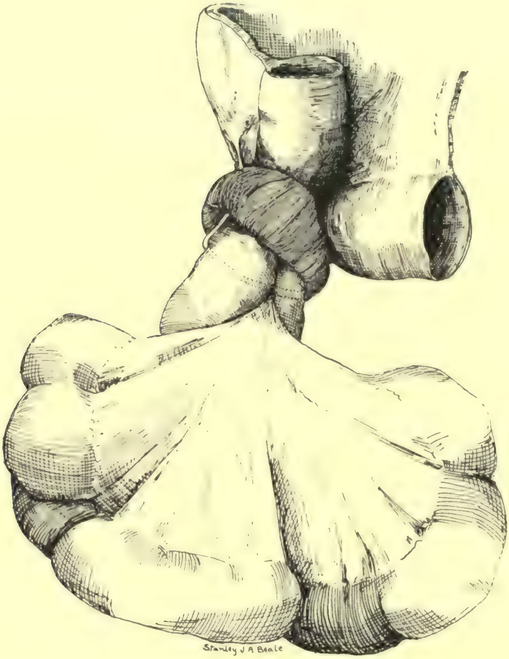


FIG. 35.—TO ILLUSTRATE THE CONDITION FOUND IN CASE II.

CASE I.—Incarceration under a Meckel's diverticulum probably attached to omentum in the right upper part of the abdomen. Resection of the cord; recovery.

William C., aged sixteen, was admitted to the London Hospital in September, 1900. He had three years previously suffered from an acute abdominal attack, which lasted one week. The present attack had lasted ten days, and was of gradual onset. He had been absolutely constipated seven days, and had vomited every day for a similar time. The vomit had latterly been green, but never fæcal. His abdomen was a little distended, and coils of peristalting small gut could be seen, proving that the obstruction was partial. The abdominal wall was soft, free from tenderness, and moved well on respiration. An obscure resistance could be felt in the right loin at intervals. His temperature was normal; his pulse 120, but regular and of good volume. Rectal examination revealed nothing. The abdomen was opened by a median laparotomy. Large hypertrophied coils of gut presented. The cæcum and lower part of the ileum were collapsed. This was one of my early cases, and I traced the distended gut downwards until it hitched. A finger then passed down the mesentery encountered a long, slack band crossing the ileum. This band was divided between ligatures. The upper end passed out of sight high up into the right side of the abdomen, where it was apparently attached to omentum. It was pulled down as far as possible, ligatured, and cut off. The lower end was then followed down. It terminated in the apex of a short, thimble-like diverticulum springing from the free margin of a lower coil of ileum. The band was cut short after ligature at the apex of the short diverticle. I found but one line of constriction on the gut, where distended intestine joined collapsed. This line was in good condition.

The boy made a good recovery except for an attack of left renal colic ten days after operation. He passed flatus the morning after operation, and his bowels were open later in the same day.

CASE II.—Acute strangulation of a Meckel's diverticulum by its own fibrous extremity; gangrene. Three feet of ileum above the diverticulum was also incarcerated in the noose.

Ernest A., aged twenty-six, was admitted to the London Hospital in July, 1902. He had suffered from no previous abdominal disease. Two days previously he had suddenly been seized with severe abdominal pain referred to the navel. He had vomited ever since, and had passed neither fæces nor flatus since the onset of the pain.

For one day he had suffered from persistent hiccough. The patient was collapsed, his eyes were ringed; his pulse 84, but very feeble; his temperature 100·5°, and his tongue dry and foul. The abdomen was rigid and tender, especially below the umbilicus. It was not distended, and no tumour could be made out. A turpentine enema was retained.

On opening the abdomen much turbid and blood-stained fluid was found free in the peritoneum. The wound was filled with distended and congested coils of gut. A hard, tense, and black mass was encountered on the right side of the abdomen, and was drawn into the wound. It was found to be a gangrenous Meckel's diverticulum encircling the 3 feet of ileum immediately above it, and twisted so that its cordlike extremity crossed and strangled its own base and the adjacent part of the ileum. The end of the cord was attached to the mesentery about 1 inch below the diverticulum. The arrangement of the strangulation is shown in

Fig. 35. The cord was double-clamped and divided and the diverticulum unwound. On examining the parts, I was of opinion that the 3 feet of ileum which had been in the band, and which was purple but glistening, would recover. The base of the diverticulum and the adjacent part of the ileum where the fibrous cord had crossed it was obviously green and gangrenous. I accordingly resected about 3 inches of the ileum, including the root of the diverticle, and performed end-to-end anastomosis. This was very difficult, because the mesentery was so short at this point. I could not be certain whether this shortening was a congenital deformity, or was the natural length of the mesentery 2 feet from the ileo-cæcal valve; but combined with the distended gut it made the operation tedious and unduly long.

Progress.—He lived twenty-four hours, and during this time he had one large and several small motions. He was, however, profoundly collapsed and restless. He did not vomit. At the autopsy it was found that the 3 feet of ileum which had been strangled in the band had passed on to gangrene. This coil was plum-coloured and covered with patches of lymph. General peritonitis was present. The suture-line was water-tight. The anastomosis was 2 feet above the ileo-cæcal valve. Apparently the whole coil of ileum should have been resected, but I do not think the patient would have recovered, as he had general peritonitis when admitted to hospital.

CASE III.—Incarceration of a coil of gut beneath a Meckel's diverticulum attached to the mesentery; partial obstruction; volvulus of the diverticulum. Death during delivery of intestines.

One evening in September, 1904, I was asked to see, in consultation in the country, a boy aged ten. His illness had commenced somewhat suddenly seven days before. He had suffered from attacks of colicky pain, but pain had not been a prominent symptom. He had passed no motion since the attack commenced, but flatus had been evacuated for the first three or four days. Distension was well marked on the fourth day, and peristalting coils of gut were visible. On this day he was seen by a consulting physician, who, considering his temperature and pulse were normal and his pain not severe, thought he was suffering from constipation, and prescribed two consecutive ounces of castor-oil. He did not think it a case for surgical interference. The vomiting stopped on the fifth day, but the bowels remained absolutely constipated and the distension increased. Symptoms of toxic poisoning appeared, and his temperature fell to 95° F., and his pulse to 75. When I saw him at about 9.30 p.m. on the seventh day of his illness, his eyes were sunken, a hæmorrhagic toxic rash had appeared over his abdomen and chest, his belly was greatly distended, and slowly peristalting coils of gut were visible. A diagnosis was made of incarceration below a slack band, either a Meckel's diverticulum or a solitary peritoneal band.

I had not my instruments with me, and had to return to town for them. The operation was performed the next morning. All preparations were made for delivering the hugely distended coils of gut out of the wound. The room was heated as much as possible, and many hot sterilized towels were prepared. When the abdomen was opened, hugely hypertrophied and congested coils of gut at once escaped. They were rapidly delivered and wrapped up in the hot towels. Nearly the whole of the small intestine had to be so dealt with before the cause of the obstruction could be reached. It was a Meckel's diverticulum, the free end of which was apparently attached to the under-surface of the adjacent mesentery; about 18 inches of ileum above the diverticle had passed beneath the arcade so formed, and had rolled the diverticulum over three times, producing volvulus of both ends and gangrene (*vide* Fig. 36). The diverticulum was 18 inches from the ileo-cæcal valve, and this coil was collapsed. The whole area of strangulation was packed away at the root of the mesentery, and covered by the greatly distended coils above. The fibrous extremity of the diverticle was double-clamped and divided. The incarcerated coil seemed in fair condition. The diverticulum was untwisted, and it was then found that the ileum was much constricted at this point, apparently by long-continued traction, and was not more than ½ inch in diameter.

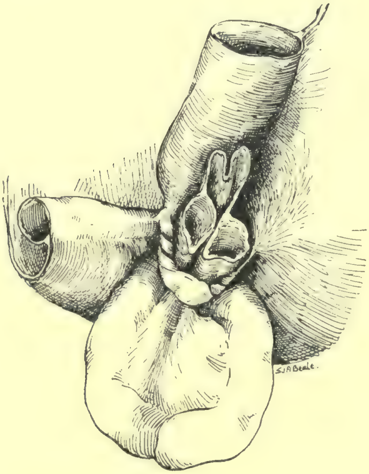


Fig. 36.

The urgency of the case did not admit of a formal resection. I accordingly crushed the narrow base of the diverticle, ligatured it with catgut, and was inserting a purse-string suture for invagination, when the boy died. The operation had so far taken some ten minutes. His pulse had failed rapidly since his intestines were delivered, and no anæsthetic had been given since then; but it was impossible to reach the diverticle, from its position, until the intestines had been got out of the way.

The obstruction in this case was also composite and cumulative. Part of it was in the incarcerated coil of ileum, and part at the strictured attachment of the diverticulum.

(5) *A Stricture of the Ileum exists in Connection with a Meckel's Diverticulum.*—Strictures are not infrequent at the point of origin of a Meckel's diverticulum, and are usually just above it. There appear to be several varieties, due to differing causes.

(i.) *Those of Congenital Origin.*—The vitelline canal is normally removed by an obliterative process, and it can be readily understood that this may be irregular in its distribution, especially where it is directed to closing the orifice of communication between the vitelline duct and the ileum. It may then produce stenosis of the ileum. It is curious, however, that such strictures are always above the diverticulum, as far as the specimens I have seen show, for it is not clear why the obliterative process should not affect the gut below as often as above the attachment of the diverticle. Such a stricture is shown in Fig. 31. The gut immediately above the root of the diverticulum was only the size of a lead-pencil for a distance of 1 inch, when it suddenly increased to dimensions even larger than normal. The stricture does not seem to have produced much trouble until the diverticulum became inverted and led to an enteric intussusception.

In Fig. 37 two strictures were present, but we shall only refer here to the one immediately above the diverticulum. The specimen illustrated here was obtained from a child four days old, whose adherent diverticulum had never left a congenital umbilical hernia to the apex of which it was adherent, yet the gut was greatly narrowed at this point.

Traction can scarcely have played a part in this case, although at the time of operation the gut was certainly kinked at this point, apparently by the band formed by the mesentery of the diverticulum.

The origin of these stenoses from irregular obliteration of the root of a Meckel's diverticulum is supported by the fact that two further degrees of the same process are found at this same spot in the ileum. In the first the lumen is occluded by a septum formed of mucous membrane only (*vide* Leopold Hudson, "On Congenital Abnormalities of the Lower Ileum," Transactions of the Pathological Society, vol. xl., p. 98, February, 1889). In the more complete form there is a break in the continuity of the ileum. In neither of these degrees is a diverticulum present.

(ii.) *Traction Strictures.*—Sir Frederick Treves fully discussed these strictures in his work on Intestinal Obstruction. He there states that he was at one time inclined to regard these strictures as acquired and due to the effects of long-continued traction on the gut. These strictures are usually found in connection with adherent diverticula, and the tension and kinking might well lead to transient attacks of obstruction, and so to ulceration and cicatricial contraction. He adds, however, that an examination of specimens showed no evidence of recent ulcers nor the scars of old ones. Moreover, Fig. 31 shows that such a stricture may exist above a diverticulum so free that it could become inverted. There is no need, however, to invoke the aid of ulceration. The intestine is of so plastic a character that any long-continued tension might well draw it out and stenose its lumen. As Sir Frederick Treves points out, a similar stenosis is found wherever

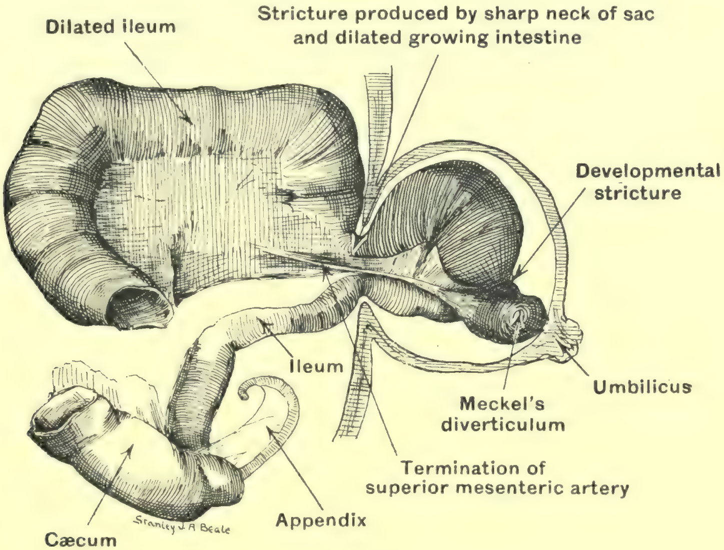

Dilated ileum
Stricture produced by sharp neck of sac and dilated growing intestine
Developmental stricture
Ileum
Umbilicus
Meckel's diverticulum
Termination of superior mesenteric artery
Appendix
Cæcum
Stanley J A Beale

FIG. 37.

any peritoneal band or adhesion drags on the small intestine, and this where there is no suggestion that stricture and band alike originated from a pre-existing ulcer. When the diverticle is adherent, the tension would naturally fall on its upper attachment, for the numerous coils above would become distended and heavy with accumulated fluid, whilst those below would not only be short, but would rapidly empty into the cæcum.

It would therefore appear that some of these strictures are congenital in origin and some are acquired by traction, but it is often difficult to say in any given case which was the determining cause.

(iii.) *The mesentery of a Meckel's diverticulum* is often well developed, and may constrict or kink the adjacent coil of ileum so as to lead to a secondary stenosis. Fig. 23 illustrates this point, and needs no further description here.

(iv.) *The neck of a congenital umbilical hernia may stricture the coil of ileum above a diverticulum so as nearly to divide it.* A patient was admitted to the London Hospital when four days old, with a congenital strangulated umbilical hernia. I resected the sac with very little anæsthetic, reduced the coil of ileum and diverticulum, and sutured the neck of the sac. The diverticulum was adherent to the umbilical scar at the apex of the sac. The child died next day, and the specimen shown in Fig. 37 was obtained, and is now in the London Hospital Museum.

In addition to the developmental stricture immediately above the diverticulum, there is another some 2 inches higher up the ileum. At the operation the sharp edge of the neck of the sac was found in this furrow, which nearly divided the gut. The entire specimen is a beautiful example of the persistence of that stage of umbilical development at which a funnel-shaped sac exists in the root of the cord. The vitelline canal did not, however, break free from the umbilicus. The ileum continued to grow, whilst its upper coil became distended with meconium. Simultaneously the ring constricted in an attempt to close the umbilical orifice. The consequence of these two processes was a very narrow stenosis. Had the diverticulum separated at some time prior to birth, and the coil of gut become reduced, the stricture might have been found at some subsequent date and attributed to developmental obliteration. It must be admitted, however, that this stenosis is not immediately above the diverticulum, a spot which is occupied by another and undoubtedly developmental stricture.

There are usually no symptoms from these strictures. A stricture of the ileum must be exceedingly narrow to offer any serious obstruction to its fluid contents. In several instances ulceration has occurred at the site of stenosis, and general peritonitis has followed perforation.

The majority of the specimens have, however, been obtained from cases in which the diverticulum led to snaring or became invaginated, as in Fig. 23.

(6) *The Meckel's Diverticulum has entirely disappeared, but the Vitello-Intestinal Vessels have persisted as a Fibrous Cord.*—An example of this condition is shown in Fig. 38. My attention was drawn to it, in the body of an old woman, by a student in the dissecting-room when I was a demonstrator, and the specimen is now in the museum of the London Hospital. It will be seen that a fibrous cord springs from the umbilicus, and passes, not to the free border of the ileum, but over it to join the mesentery, where it becomes continuous with a main branch of the superior mesenteric artery, which is the persistent portion of the vitelline artery. In this instance the band had constricted the gut beneath it, but there was no evidence of obstruction. Such bands might, of course, lead to any of the varieties of strangulation which have been discussed under other varieties of bands.

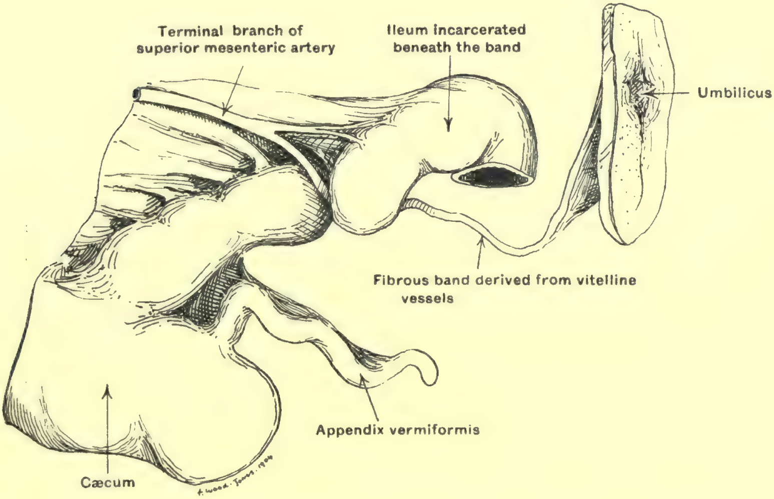

Terminal branch of superior mesenteric artery
Ileum incarcerated beneath the band
Umbilicus
Fibrous band derived from vitelline vessels
Appendix vermiformis
Cæcum

FIG. 38.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM.

Cases have been described where such cords have become detached from the umbilicus, and have remained free at one end or have formed some other attachment. They are also said sometimes to separate at their mesenteric end. The close association should be noted between these vascular bands and those cases already alluded to, where a Meckel's diverticulum chiefly caused trouble on account of the attachment of its mesentery.

Other Diverticula of the Alimentary Canal.—Diverticula occur at almost any point along the alimentary canal from the œsophagus to the rectum, but it would be scarcely relevant to treat of them at great length here, as they seldom obstruct the bowel. In a few cases, however, they become so large as to press upon the gut above or below their entrance into it, or, by traction at their point of attachment, stricture or kink the intestine. Pathologically they may be classified as follows :

(i.) *Pouches due to Traction.*—Some structure, more usually a lymphatic gland, becomes inflamed, and adherent to some neighbouring part of the alimentary canal. When resolution of the inflammation occurs, followed by cicatricial contraction, a pouch is drawn out from the alimentary canal. Such pouches are found in the œsophagus opposite the glands below the bifurcation of the trachea. In the duodenum, where it surrounds the head of the pancreas; and perhaps this explains those diverticula which are sometimes found projecting from the small intestine between the layers of the mesentery. A pouch is sometimes found in the base of an adherent appendix epiploica, or even of a peritoneal band or adhesion, and this must be regarded as a traction pouch.

(ii.) *Pouches due to Distension.*—As the bladder may become pouched when an enlarged prostate or stricture is present, so the entire colon is sometimes found studded with diverticula in cases of chronic constipation. These pouches are formed by hernia of the mucous membrane through the muscular coat between the muscular bands, which are often greatly hypertrophied. They are often innumerable, and are sometimes filled with fæcal concretions. The colon is bound down by chronic peritonitis around them. They are, however, rather a consequence than a cause of obstruction.

(iii.) When *foreign bodies* become lodged at some point along the alimentary canal, they sometimes develop a pouch, enclosed in which they no longer obstruct the direct stream.

(iv.) *Chronic abscesses* which burst into the canal are said in a few cases to become lined with epithelium, and in this way give rise to diverticula.

(v.) *Aberrant diverticula*, especially when they are on the free border of the gut and have a well-formed muscular wall, are probably developmental in origin. Some regard them as greatly displaced Meckel's diverticula. They may occur at any point along the small intestine or colon between the entrance of the common bile-duct and the splenic flexure of the colon.

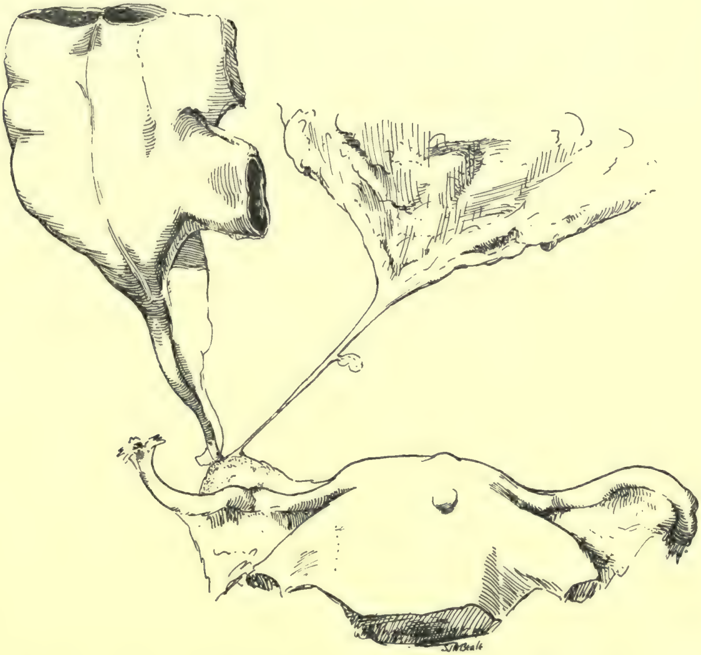


FIG. 39.—TO ILLUSTRATE THE APPENDIX VERMIFORMIS ATTACHED TO THE RIGHT OVARY.

An omental band is also present.

4. *Bands produced from Pedicles and Normal Cordlike Structures abnormally attached.*—I have no personal experience of such cases, and will therefore treat the subject shortly.

The pedicle of an ovarian tumour or of a fibroid, when it is sufficiently long, may strangle gut.

The normal organs which may produce the same results are the appendix (*vide* Fig. 39), the Fallopian tube, the hydatid of

Morgagni, and an appendix epiploica. At least one case has been recorded hard of credence, where a free appendix had snared a coil of gut. As a rule, however, the distal end of the normal organ has been found attached. A preliminary stage appears to be gone through in most cases in which the constant abdominal movements draw out the attached structure to an extraordinary length. I have seen a specimen of a Fallopian tube which thus became some 5 inches in length (*vide* Fig. 40).

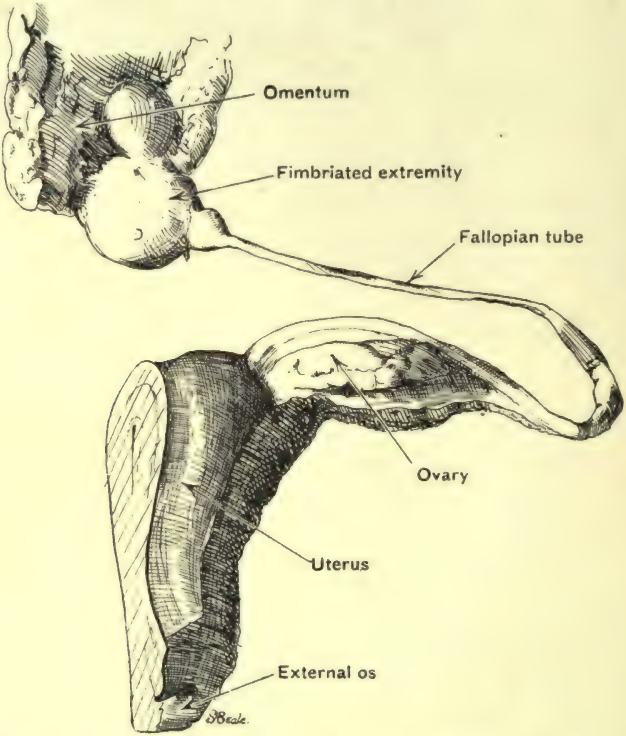


FIG. 40.—FROM A SPECIMEN IN THE LONDON HOSPITAL MUSEUM.

Strangulation by knotting, noosing, or beneath or over the cord, may occur just as with any other form of band. Mr. James Berry has told me of a case of this kind which occurred at St. Bartholomew's Hospital, in which the apex of two appendices epiploicæ became adherent to one another, and through the loop so formed a coil of small intestine passed. The mesentery may become so attached that a coil of gut may become incarcerated between it and the abdominal wall.

(B) **Strangulation through Slits, Apertures, or Holes.**—The great majority of these cases involve the mesentery or the omentum, and are congenital or traumatic in origin. A few are due to atrophy.

Seven cases of apertures have occurred at the London Hospital in thirteen years, out of 669 cases of intestinal obstruction: 5 were men, 2 women; 6 died and 1 survived. The apertures were situated as follows: Mesentery, 3; ascending mesocolon, 1; omentum, 1; small omentum, 1; 1 stab wound of peritoneum, through which gut passed into subperitoneal tissues.

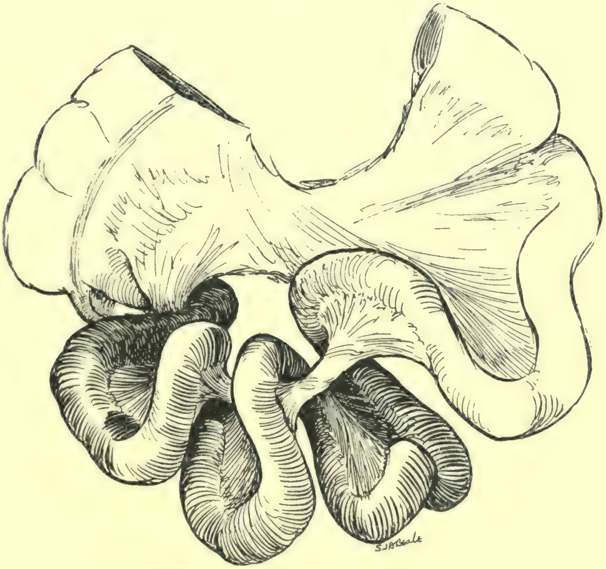


Fig. 41.—To illustrate Strangulation through an Aperture in the Mesentery.

The *mesentery* is most frequently involved, and in its lower part near the cæcum (*vide* Fig. 41). Sir Frederick Treves has described a spot in this situation free from fat lymphatics and vessels, and surrounded by the anastomotic branch between the ileo-colic and the last mesenteric vessel. This spot is always thin, and may be actually perforated (" Hunterian Lectures on the Anatomy of the Intestinal Canal and Peritoneum," London, 1885, p. 28). Strangulation through such an aperture has been recorded. In most cases some previous trauma has occurred, such as a kick or the passage of a wheel over the abdomen. I have added such a specimen to the London Hospital Museum, where a large aperture had been produced in the mesentery of the lower ileum, but death was due to subsequent stricture of the intestine at two adjacent spots, where the gut had been nearly completely ruptured. In the case of another specimen, in the museum of the Royal Free Hospital, a surgeon whilst exploring the abdomen had in all probability passed his finger through the fatty, friable mesentery. Two coils of gut had become incarcerated in the aperture, one from before back, and the other from behind forwards.

Strangulation has also been recorded through slits in the mesentery of the transverse and descending colon, and even of the appendix.

The *omentum* is the second commonest site of apertures (*vide* Fig. 21). The majority have followed injury, but some have apparently been congenital in origin. After peritonitis the omentum may be found divided into many bands, with apertures between (*vide* Fig. 20). In other cases nearly the whole of the small intestine has protruded through a large aperture in the omentum, which was reduced to a cordlike sling below it. Apertures in the adherent omentum have frequently been produced by surgeons in order to reach structures behind it. This is particularly likely to occur when operating on the pelvic viscera in women.

Less common sites for apertures are in broad membranous adhesions, in the suspensory ligament of the liver or spleen, in the broad ligament of the uterus, and in the base of flat appendices epiploicæ.

Slits have also been produced by the adhesion of one organ to another, such as intestine to intestine, or intestine to the uterus (*vide* Fig. 42), the broad ligament, or the abdominal wall, especially at the hernial rings. False ligaments may be stretched parallel to such structures as the broad ligament or the utero-sacral ligament, a slit intervening.

Coils of gut have become incarcerated in rents in the bladder, uterus, or rectum, or even in a coil of gut which had been transfixed. Finally, in wounds of the abdominal wall, intestine may pass through the slit in the peritoneum and become incarcerated in the subperitoneal tissues.

The *Symptoms* are nearly always those of the most acute form of intestinal obstruction. In only one case at the London Hospital were the symptoms subacute, and contracting coils of intestine were visible whilst flatus was passed. In this case nearly the whole of the small intestine had passed through a very large aperture in the omentum. This was the single case of obstruction due to aperture which has recovered at the London Hospital in thirteen years.

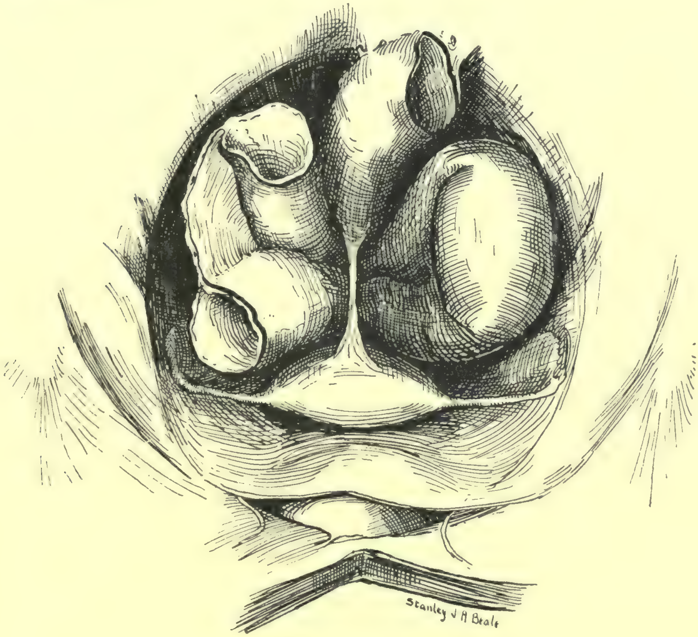


FIG. 42.—SHOWING STRANGULATION UNDER A BAND PRODUCED BY ADHESION OF UTERUS TO RECTUM.

The *Treatment* is to enlarge the aperture and reduce the strangulated intestine, which must be dealt with on its merits. The aperture should then be sewn up.

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Treves, Sir Frederick, Hunterian Lectures on the Anatomy of the Intestinal Canal and Peritoneum, London, 1885, p. 28.

(C) **Strangulation by Internal Hernia.**—For practical purposes it would be well to include here all those cases of hernia in which symptoms of strangulation or incarceration are present, but no tumour is visible externally. It is customary, however, to describe a number of such cases as external herniæ. For the sake of completeness these will be enumerated here, but not described.

External Herniæ without an External Tumour in which Strangulation may be produced.—1. Exceedingly small external herniæ through the femoral or inguinal rings, the linea alba, lineæ transversæ or semilunaris, or Gimbernat's ligament in fat people.

2. Interstitial herniæ at the inguinal, femoral, or umbilical rings. These may be present with or without an ordinary hernia at the same orifice.

3. Obturator, ischiatic, perineal, pudendal, rectal, vaginal, and lumbar herniæ, in all of which the external tumour is easily overlooked.

Obturator hernia is the only one of these that at all commonly causes trouble. Its symptoms are as follows: Fulness and tenderness on deep palpation over the inner part of Scarpa's triangle. Pain, formication, and rigidity, down the inner side of the thigh. It is four times as common in women as men, and the patients are nearly all over forty years of age. One case has occurred at the London Hospital in thirteen years. It was not diagnosed. It was reduced by laparotomy. The woman died from paralysis of the gut two days later.

True Internal Herniæ.—Thirteen cases of true internal hernia have been admitted to the surgical wards of the London Hospital in thirteen years, out of 669 cases of obstruction. Four were prevesical, three diaphragmatic, two duodeno-jejunal, two pericæcal, one into Douglas's pouch (the mouth of which was constricted by adhesions), one in the left sigmoid fossa in a pouch formed by adhesions between the lower surface of the mesosigmoid and the iliac fossa.

True internal herniæ are as follows:

1. *Subperitoneal Herniæ in the Situation of the Inguinal Ring.*—Hernial sacs are found projecting outwards (hernia intra-iliaca), downwards (hernia retropubica), or inwards (hernia antevesicalis) from the site of the internal inguinal ring. They are situated in the subperitoneal tissues. Some are associated with ordinary inguinal sacs. Some cases give a history of reduction *en masse*. It has been inferred, therefore, by some authorities that all such sacs are secondary to ordinary inguinal

hernias either by repeated reduction producing pouching (hernia *en bissac*) or by reduction *en masse*. In the case of antevesical hernia, at least, the sac may open in the middle line; the patient may never have had an inguinal hernia, and neither ring may be enlarged. Four cases of antevesical hernia have occurred at the London Hospital in thirteen years: one under the care of Mr. Jonathan Hutchinson, junior, and three under that of the writer. All recovered. One was a congenital sac in the middle line, two were examples of reduction *en masse*, and one of hernia *en bissac*.

2. *Hernia Interna Vaginalis Testiculi*.—In this form of hernia the testicle and vaginal pouch of peritoneum have failed to traverse the inguinal canal. The pouch is subperitoneal, and lies on the psoas or iliacus muscle. The testis is, of course, absent from the scrotum.

3. *Hernia Iliaco-Subfacialis*.—This hernia was first described by Biesiadecki. The orifice of the sac is in either iliac fossa. The sac itself passes through the iliac fascia and lies in contact with the iliacus muscle. Such a case was operated on by Mr. Mansell Moullin in the London Hospital. The specimen is now in the museum, and shows the intimate relation of the external iliac artery to the sac. The pouch is said by some to originate from the ridge formed by the insertion of the psoas minor into the iliac fascia; by others it is attributed to the tense margin of the strong lower portion of the iliac fascia.

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Biesiadecki, Unters aus d. Path. Anat. Inst. in Krakan Wien, 1872, p. 19 *et seq*.

4. *Hernia Duodeno-Jejunalis.*—In the dead body, if the great omentum and transverse colon be drawn upward and the small intestines held over to the right, the duodeno-jejunal flexure will, in the great majority of cases, be seen to be surrounded on the left by a crescentic fold, the plica duodeno-jejunalis. Between the fold and the flexure the fossa duodeno-jejunalis is usually present.

Etiology.—The duodeno-jejunal fossa is probably always developmental in origin. In practically all cases which have produced a left hernial sac, the neck of that sac has been found to contain, upon the left side and above, the inferior mesenteric vein passing to its ending in the splenic vein, and, to the left and below, the left colic branch of the inferior mesenteric artery. In right duodenal hernia the neck of the sack is surrounded by the superior mesenteric artery or its continuation, the ileo-colic. The origin of these herniæ would appear to be due, therefore, to the vascular fold or mesentery thrown up by these vessels in the peritoneum of the posterior abdominal wall (Waldeyer). Many other folds and pouches have been described by anatomists, even to the number of nine (Moynihan), but they seldom if ever produce hernial sacs. They have been attributed to traction by Treitz, during the development of the colonic loop and the duodeno-jejunal flexure; to fusion of the left side of the duodenum and the descending mesocolon by Moynihan; and to the persistence of part of the mesoduodenum by Treves.

It is as a rule the first part of the jejunum which is contained in the pouch, and by its continuous movements enlarges it, drawing in more and more of the gut. The sac spreads in the retroperitoneal tissues in the direction of least resistance. As a rule it occupies the left side of the abdomen, surrounded by the transverse and descending colon, and with its orifice directed towards the right. When the pouch passes upwards, it will lie in the root of the transverse mesocolon in front of the pancreas and left kidney, and covered in front by the transverse colon and the stomach. The orifice will then be directed downwards and towards the right (hernia mesocolica). When the hernia has extended into the right side of the abdomen, it will pass below the arch formed by the superior mesenteric artery, and be surrounded by the ascending and transverse colon; or it may pass even farther to the right beyond the ascending colon, so that the later structure lies in front of the sac, as in Brösike's case.

As the hernia enlarges, the duodeno-jejunal flexure is as a rule drawn into the posterior wall of the sac, so that only the returning coil is seen passing out of the orifice, and strangulation is avoided. In this way, as in Neubauer's original case (1776), the whole of the small intestine may come to lie in the sac, and only the terminal coil of ileum pass through the orifice to enter the cæcum.

In rare instances the upper part of the jejunum may be contained in the sac, but in addition a coil of the lower ileum may enter the orifice and become strangulated, as in a case encountered by the writer.

Symptoms.—Strangulation is the exception in duodeno jejunal herniæ. It occurred but three times in forty-two cases collected by Leichtenstern. Two cases have occurred at the London Hospital in the last thirteen years; one was incarcerated, the other strangulated. The symptoms are those due to tension produced by the sac on the peritoneum, mesenteries, stomach, colon, and inferior mesenteric vein. They are: colic, dyspepsia, constipation and irregularity of the bowels, abdominal unrest, depression of spirits, and piles, which bleed freely. The physical signs are of a circumscribed globular swelling occupying the epigastrium and left side of the abdomen, and resembling a movable cyst; but the tumour is always resonant on percussion, and borborygmi are heard on auscultation.

Treatment.—The abdomen should be opened and the gut reduced. The neck of the sac should then be sewn up. The sac might be drained posteriorly. It should be remembered that the neck of the sac contains as a rule the greatly dilated inferior mesenteric vein and the left colic artery, which must not be divided when incising the neck of the sac.

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5. *Internal Herniæ around the Cæcum and Appendix.*—Two cases of pericæcal hernia have occurred at the London Hospital among 669 cases of intestinal obstruction in thirteen years; both were strangulated and both died. Neither was properly dissected.

Anatomy.—Three folds of peritoneum are described in this region, and five pouches.

(i.) *Folds.*—(1) *The anterior vascular or ileo-cæcal fold* conveys a terminal branch of the ileo-colic artery in front of the end of the ileum to the cæcum at the root of the appendix.

(2) *The ileo-appendicular or bloodless fold of Treves* fills in the angle between the end of the ileum and the cæcum and root of the appendix. It contains muscular fibres continuous with those of the cæcum, and in its margin a recurrent branch of the appendicular artery to the ileum.

(3) *The meso-apppendix* passes from the mesentery behind the ileum to the proximal half of the appendix. It contains in its free border the appendicular branches of the ileo-colic vessels.

(ii.) *Cæcal Pouches.*—(1) *The ileo-colic fossa,* when present, lies between folds (1) and (2).

(2) *The ileo-cæcal fossa* lies between folds (2) and (3). According to Moynihan, four cases of hernia into this fossa have been recorded by Tuffier, Little, and Partridge. In Tuffier's case there was a hernial sac. In those of Little and Partridge the intestine passed through a slit in connection with the meso-appendix, and no sac was present.

(3) *The retro-appendicular fossa* lies behind the appendix and its mesentery. In one case recorded by Snow, strangulation of the ileum appears to have occurred in this fossa.

(4) *The retrocæcal or retrocolic fossa* is usually seen when the cæcum is drawn up. It is formed by incomplete fusion of the mesentery of the primitive cæcum and ascending colon to the right loin.

Several cases of retrocolic hernia have been recorded, but much doubt has been raised as to whether they were really into this fossa. The appendix is sometimes found in this fossa, the mouth of which may be closed (Lockwood).

(5) *The infracæcal pouch* lies in the right iliac fossa just below the cæcum. Its mouth looks upwards. Probably it is identical with the hernia iliaco-subfacialis already described. A careful dissection of so-called retrocæcal hernias would probably show that many of them were in reality into this fossa.

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6. *Hernia Intersigmoidea.*—The fossa sigmoidea was first mentioned by Hensing in 1742. The frequency of its occurrence has been variously estimated as from 52 per cent. (Gruber) to 84 per cent. (Treves). It is almost constant in babies, but is obliterated, as life goes on, by the chronic inflammatory changes in the mesosigmoid.

The origin of this fossa has been attributed to two vascular folds of peritoneum thrown up by the sigmoid artery (Waldeyer, Treves). Moynihan regards it as due to imperfect fusion of the primitive mesentery of the descending colon with the peritoneum of the left loin. The orifice of the fossa lies on the under-surface of the root of the mesosigmoid, close to the inner border of the psoas magnus. The sigmoid vessels are found in the anterior margin of the orifice. Behind the posterior wall of the fossa lie the common iliac artery at or near its bifurcation, and the ureter. The depth of the fossa varies from a mere dimple to 3 inches. Its apex may reach to the middle of the kidney or the pancreas.

Hernia into this fossa is extraordinarily rare. Leichtenstern only mentions one case (Lawrence). Mr. F. S. Eve has described an undoubted case; the rest are doubtful.

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7. *Hernia Intra-epiploica.*—Coils of intestine have been found surrounded by a sac of omentum; they are probably previously reduced inguinal herniæ.

8. *Hernia Ligamenti Uteri Lati.*—Small pouches are sometimes found projecting into the substance of the broad ligament of the uterus. Such a specimen is in the museum of the London Hospital. A partial enterocele of the ileum had occurred into the pouch.

9. *Hernia into Douglas's Pouch.*—The writer has operated on such a case where the mouth of the pouch was constricted by adhesions between the uterus and rectum. The adhesions were divided and the ileum released. The patient recovered. Another case has recently (1906) occurred at the London Hospital. The patient refused operation and died.

10. *Hernia through the Foramen of Winslow into the Lesser Sac of the Peritoneum.*—Eight cases of this hernia have been collected by Moynihan. In six of these the small intestine was involved, and in two the colon; seven were men, and one a woman. Their ages ranged from twenty-five to forty-four years. It would appear that gross congenital abnormality in the attachment of the colon is necessary for its occurrence; otherwise the transverse

colon and its mesentery effectually intervene between the small intestine and the foramen. The symptoms are those of acute strangulation, with severe pain in the epigastrium and a swelling resonant on deep percussion. In most cases the gut cannot be reduced, and enterostomy would therefore appear to be the treatment. No recorded case has survived.

11. *Hernia Diaphragmatica.*—This is stated to be the most common form of internal hernia, yet it is very rare, for it has been found but three times in thirteen years at the London Hospital, in 669 cases of intestinal obstruction, and in one of these the obstruction was due to another cause.

Leichtenstern has compiled a most complete account of this form of hernia based on 252 cases (1877); and according to Nothnagel later authorities are Grosser, 433 cases up to 1899, and Struppler, 500 cases up to 1901.

Anatomy.—The hernia is said to be *false* when only an orifice exists in the diaphragm and the contents lie bare in the thoracic cavity, and *true* when a sac of peritoneum exists which may or may not be covered by pleura. Leichtenstern gives the proportion as 212 cases of false hernias to 28 cases of true.

The position of the liver renders this hernia much less frequent on the right side. Leichtenstern found 180 cases were on the left side, and only 37 on the right. The orifice in the diaphragm is due to either a congenital deficiency or to some traumatism, such as may result from stabs, falls, or crushes, as when the patient has been run over. When the symptoms of diaphragmatic hernia have followed severe vomiting or the violent expulsive efforts of parturition, it is probable that the orifice in the diaphragm already existed.

The acquired form is much more common in men (Leichtenstern, 128 men to 22 women) and in those who follow laborious or dangerous occupations, such as sailors, soldiers, carpenters, and slaters.

The *aperture* may be small, or one-half the diaphragm may be missing. The hole is usually in the muscular part of the diaphragm, and is more often behind (*vide* Fig. 43).

Certain spots are characteristic, such as (1) where the œsophagus pierces the diaphragm; (2) where a gap exists between the fibres passing to the ensiform process and those to the cartilage of the seventh rib; (3) at a similar gap behind, between attachments to the lumbar spine and the twelfth rib; and (4) where the sympathetic nerve pierces the crus of the diaphragm.

In no case, apparently, has a hernia passed through the orifices for the vena cava or aorta. In most cases the hernia enters the left pleura, but less often it is found in the anterior or posterior mediastinum, or even in the right pleura.

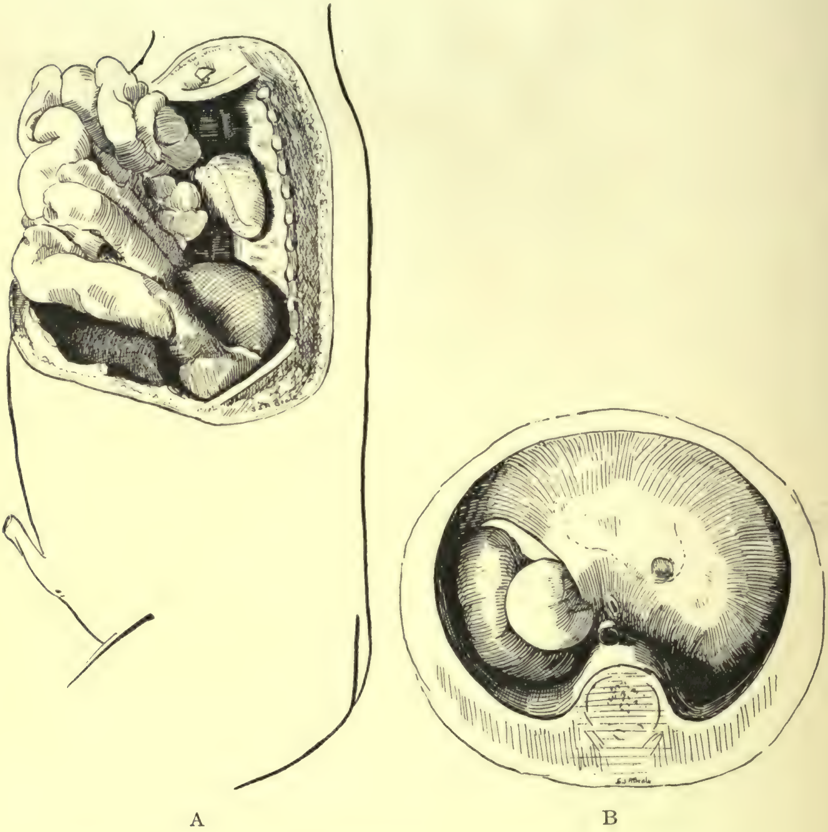

Fig. 43.—From a Case of Diaphragmatic Hernia in a Child who was Born Dead.

A, before reduction. B, in opening in the diaphragm as seen from above after partial reduction.

The contents are nearly always the stomach, and then in order of frequency the transverse colon, the omentum, the small intestine, the spleen, left lobe of liver, the pancreas, and left kidney. It is always the greater curve of the stomach which enters the orifice, so that the organ is rotated on its orifices and lesser curve.

Symptoms.—A baby the subject of diaphragmatic hernia may be born dead. On the other hand, it may live for many years with more or less definite symptoms of thoracic and abdominal disease. The writer heard recently of such a case in a children's hospital, where the left pleura was opened for a supposed empyema, whereupon intestine presented in the wound. In traumatic cases the symptoms may supervene directly after the injury, but even then they may subside and become chronic, so that life is prolonged for years. On the other hand, as in a stabbing case of Mr. Hugh M. Rigby's at the London Hospital, a long interval may elapse between the injury and the strangulation of viscera in the orifice. In its chronic form diaphragmatic hernia can be diagnosed in favourable cases on careful and repeated examinations. Leichtenstern states that a correct diagnosis was made five times in 252 cases.

The thoracic signs are almost identical with those of pneumothorax. In both conditions the chest is prominent and fixed, and the heart is displaced. On auscultation breath-sounds are absent, and vocal fremitus is not obtained in either condition. On percussion, in both the note is drumlike and tympanitic, or is metallic in character when auscultatory percussion is employed. Succussion may also be obtained in either state. There are, however, differences. Pneumothorax involves the whole pleura, and is as often right as left; whilst diaphragmatic hernia may involve only the lower part of the pleura, and is nearly always left. In pneumothorax the signs are evenly distributed over the chest wall, and undergo but slow changes. In diaphragmatic hernia they vary very greatly at different points, and may change even during a single examination. Abdominal symptoms, such as dysphagia, dyspepsia, vomiting, or actual obstruction, are not marked in pneumothorax, whereas they are seldom absent in diaphragmatic hernia. Moreover, the thoracic signs and symptoms will be found to vary in the latter condition with changes in the abdomen, such as the taking of food, flatulent distension, or straining. Indeed, when the patient strains the abdomen may become flatter and the thorax more distended. Finally, actual borborygmi may be heard in the pleural cavity in diaphragmatic hernia. Special means may be employed to examine suspected cases. The chest may be auscultated whilst the patient swallows. The stomach and colon may be distended with fluids or with gas, and the effect on the thoracic condition noted. Gastrodiaphany may be practised, or the Röntgen rays employed after the stomach or colon have been filled with a bismuth emulsion. Death may result either from the strangulation of the abdominal viscera, or from interference with the circulation and respiration due to pressure by the incarcerated organs.

The *Treatment* when any is indicated is surgical. It is often necessary to open the thorax as well as the abdomen, in order to safely reduce the viscera and close the orifice.

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4. INTUSSUSCEPTION.

Definition.—John Hunter defined an intussusception as "the passing of one portion of the intestine into another, and it is commonly, I believe, from the upper passing into the lower."

Nomenclature.—In its simple form an intussusception is formed of three tubes, two of which are placed within the third. In vertical section it is composed of six layers, three on either side of the central canal. From within out these layers are known as (1) *the entering*, (2) *the returning*, and (3) *the ensheathing* or *receiving* layer, or *the sheath.* The two inner tubes form a projecting polypoid-like mass into the intestine below, and are known together as the *intussusceptum*, and the outer layer is then called the *intussuscipiens.* The end of the intussusceptum is *the apex.* The upper part of the intussusception, where the entering layer passes into it, is called *the neck*, and the ridge which is formed at this point by the junction of the two outer layers is known as "le bourrellet." In a *complete intussusception* the whole circumference of the gut is invaginated. An intussusception is said to be *lateral* or *partial* when one side only of the bowel is drawn as a cone within the ensheathing layer. This condition is as a rule the result of traction on the pedicle of a polypus or a swollen Peyer's patch. Partial intussusception nearly always becomes complete at a later stage.

An intussusception, like a hernia, may be *reducible* or *irreducible*, *strangulated* or *incarcerated*, *inflamed* or *gangrenous*. According to their position they are defined as *enteric*, *entero-colic*, or *colic*.

An intussusception is usually *single* and *solitary*, but in the intussusceptions of the death-agony they are more often *multiple*, and may be *ascending* as well as *descending*.

A *simple intussusception* is one composed of three tubes or six layers.

A *compound intussusception* is formed when a simple one passes as a single mass into the coil of gut below it, thus producing a *double intussusception* with five tubes or ten layers. Even *treble* and *quadruple invaginations* have been described.

Etiology.—This matter, which appears really to be a very simple one, has been enshrouded by profuse and mysterious explanations. Sir Frederick Treves gives it as his opinion that some "irregular" or "disordered" contractions of the intestine, perhaps connected with inco-ordinate action of the circular and longitudinal muscular layers of the bowel, are the primary cause of invagination. He quotes the division into invaginatio-spasmodica and invaginatio-paralytica, in which segments of the intestine which are contracted or paralyzed are supposed to become invaginated into healthy segments. Nothnagel tetanized coils of intestines with electrodes in rabbits. The contracted segment was ensheated by the normal intestine below. He also crushed a coil of intestine, and applied his electrodes to the normal gut below, whereupon the crushed portion was invaginated into the stimulated intestine. These theories, surmises, and experiments, probably have an important bearing on the invaginations of the dying, but have nothing to do with the vast majority of the intussusceptions of the living. Even for the invaginations of the death-agony I would offer a different explanation. I once opened the abdomen of a boy whose duodenum had been ruptured by the passage of a cart-wheel, and found his small intestines in a violent state of peristalsis. Waves of contraction were rapidly sweeping both up and down his bowel. When these waves met, the larger engulfed the smaller, and an invagination was produced. These invaginations were chiefly ascending. The researches of Bayliss and Starling have shown that such irregular peristalsis can only occur when Meissner's and Auerbach's plexuses are paralyzed, but the muscular wall retains its vitality; and these are exactly the conditions likely to occur just before death from meningitis and when the great abdominal plexuses have been crushed, the circumstances under which such invaginations are usually found.

For the intussusceptions of the living a much simpler and more common explanation can be given. Pathology is only perverted physiology. The normal function of the intestine is to propel its contents downwards by waves of peristalsis. As Bayliss and Starling have shown, a bolus of food excites a reflex from the mucous membrane with which it is in contact. The coil of gut above it closes in a strong ring contraction. The coil around and below it relaxes, and is drawn over it by the longitudinal fibres. In this way the bolus is moved forward and stimulates a lower segment of mucous membrane, and fresh circular fibres contract behind it, so that the movement is progressive. Should a coil of intestine become so displaced as to touch the mucous membrane of the coil below and produce stimulation, it will be grasped and passed down the canal by normal peristalsis. The etiology of intussusception thus reduces itself to a consideration of the condition under which a part of the intestine may come in contact with the inner surface of that below it and excite this normal reflex. These conditions are as follows, and include nearly all cases of intussusception in the living:

(i.) The presence of a polypus.
(ii.) The presence of a carcinoma of the intestine.
(iii.) The inversion of a diverticulum.
(iv.) Various forms of prolapse at the ileo-colic sphincter.
(v.) The presence of a swollen and hypertrophied Peyer's patch.
(vi.) Paralytic and relaxed conditions of the colon which permit of accidental invagination.
(vii.) A segment of gangrenous gut which acts as a foreign body in rare cases. Indeed, it may be stated that, if the apex of an intussusception be carefully examined, a mechanical cause for the invagination will nearly always be found there.

(i.) The cases in which *an Intussusception is due to a Polypus* are rare, but easy to understand. The size of these slow-growing tumours (adenomata, fibromata, lipomata, and myomata), and the history which is often given, in these cases, of long-standing intestinal trouble and attacks of colic, indicate that as long as the tumour is covered by normal mucous membrane it only acts as a feeble stimulant to the mucous membrane with which it is in contact. When, however, the mucous membrane is thin and adherent or actually removed by ulceration, or the polypus is composed of new growth, then the interior of the intestine is powerfully stimulated, and the intestine fiercely grasps the obstinate mass and makes violent efforts to propel it along the lumen of the bowel. The invagination is at first lateral or partial, and is opposite the attachment of the pedicle. Later the invagination is grasped as well as the polypus ; finally the whole circumference of the bowel is drawn in, the intussusception is complete (*vide* Fig. 29). A typical case was recently reported by Dr. Andrews at the Chicago Medical Society. The polypus was a fibro-adenoma, and possessed a pedicle 4 inches long, which was attached 12 inches above the ileo-cæcal valve. The intussusception was resected, but the patient died of peritonitis.

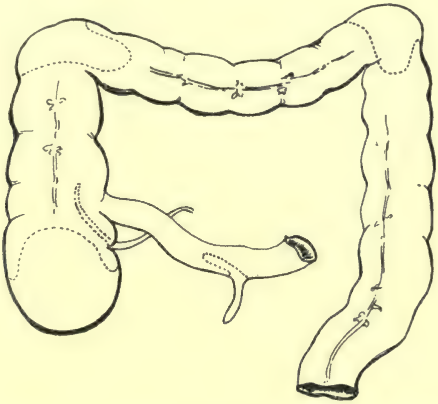

FIG. 44.—DIAGRAM TO ILLUSTRATE THE POSITION IN WHICH DIVERTICULA MAY BECOME INVERTED.

(ii.) *Carcinoma of the Colon seldom leads to Intussusception*, unless it is at the ileo-cæcal orifice. Mr. A. E. Barker has recorded a case in the upper part of the rectum, and has collected eleven others. Two such cases have occurred at the London Hospital; one, under the care of Mr. Mansell Moullin, was situated in the sigmoid or the rectum. The other, under Mr. F. S. Eve, was placed on the ileo-cæcal valve (*vide* Fig. 13). The projection of the tumour into the bowel below is partly due to growth, and partly to the distension and propulsive efforts of the bowel above, which force the stricture downward like an os uteri.

(iii.) A whole class of intussusceptions depend for their mechanical origin upon *the Inversion of Diverticula into the Intestinal Lumen* (*vide* Fig. 41). The inverted diverticulum forms a polypoid mass, and may readily excite the bowel below. The diverticula which become inverted are Meckel's diverticulum, the cæcum, the appendix, and the angles of the colon. The version of the diverticle may begin at the end by a dimpling in. This is probably a common method of origin in the cæcum, the angles of the colon, and in some cases of inversion of Meckel's diverticulum. In the appendix always, and in Meckel's diverticulum, usually the invagination commences at the base by a prolapse of the mucous membrane, probably due to spasmodic contraction of the muscular coats of the diverticulum. Many diverticles which are inverted never lead to intussusception. This is probably because they are soft, and their normal mucous covering is a feeble stimulant of peristalsis in the coil below.

An inverted Meckel's diverticulum is the point of origin of an intussusception (*vide* Fig. 30). The chief papers on this interesting subject are by Küttner, 1898; Halstead, 1902; Corner, 1903; and Sir Watson Cheyne, 1904. The latter has collected sixteen cases in his paper. As a rule the inversion commences as a prolapse of the mucous membrane into the ileum. In Hohlbeck's case the entire mucous membrane was prolapsed without inverting the diverticulum. On the other hand, the inversion may commence at the extremity by dimpling, as is shown by specimen A2718, Royal College of Surgeons Museum, where only the peripheral half of the diverticulum is inverted into the proximal half. In two cases the invagination of the apex of a free Meckel's diverticulum was due to the presence of an accessory pancreas there (Heller and Brunner).

Of twelve cases collected by Halstead, eight had gone on to intussusception, and four had remained as simple inversion of the diverticle. In two cases a stricture was associated with the diverticulum (Ewald and Cheyne). An inverted Meckel's diverticulum at first produces a lateral or partial intussusception, and only later a complete one.

The clinical history of these cases is often long and chronic, but they frequently terminate very acutely. There is a great tendency for this variety of invagination to form double and even triple intussusceptions. In Sir Watson Cheyne's case a triple invagination existed.

The diverticulum may advance to the ileo-colic sphincter, and even through it. It is then tightly strangled. When it has become fixed at the valve, a secondary ileo-cæcal intussusception is often superadded.

Cæcal intussusception: the intussusception originates in an inverted cæcum. This variety was first described by Mr. F. S. Eve in 1897. The inversion is always complete, and the fundus of the cæcum forms the apex of the intussusceptum. When such an intussusception is reduced, the cæcum remains inverted after reduction of the ileo-cæcal valve. It has usually been assumed that the inversion commences at the apex of the cæcum by some accidental dimpling in, but after carefully dissecting several specimens I am of opinion that inversion of the cæcum is usually a secondary and unimportant result of ileo-cæcal intussusception. The valve prolapses into the ascending colon and is grasped and sucked upwards. If it hangs back, and is more or less firmly attached, the loose inner wall of the cæcum, which is continuous with the lower lip of the valve, is drawn in first, and the cæcum gradually inverted from its base. The ascending colon then, having obtained a firmer grip, draws in the valve and the ileum. The proof is, that in the cases I dissected the lips of the valve were prolapsed and pouting, and it is hard to see how this condition can have been produced if the valve were passively drawn in after the cæcum.

An intussusception originates in an inverted appendix vermiformis. This condition is exceedingly rare. Mr. Herbert F. Waterhouse recorded a case in 1898, and gave notes of five others. Mr. E. M. Corner brought the literature up to 1903. Mr. Pendlebury has recently recorded another case, the specimen of which is in St. George's Hospital Museum. The inversion always commences at the base by prolapse of the mucous membrane, and it is seldom complete. In Dr. Rolleston's case a stone was fixed in the prolapsed mucous membrane, and this led him to compare this condition to that of prolapse of the ureter into the bladder when a stone is impacted there. This form of intussuspection is very chronic, and in several of the cases inversion of the appendix had not gone on to intussusception. Mr. Ewen Stabb has reported a case of intussusception in a boy of three years of age. The intussusception was caused by a hard mass in the wall of the cæcum due to an adherent appendix perforated at its base. The case recovered.

The intussusception originates in an inversion of the hepatic or splenic angles of the colon. Colonic intussusceptions occur at the hepatic and splenic flexures, and where the sigmoid flexure joins the rectum. In the case of a little girl upon whom I operated the origin of the intussusception was the dimpling in of the splenic angle into the descending colon. I have seen a similar condition at the hepatic angle.

(iv.) *Intussusception due to Various Degrees of Prolapse at the Ileo-Colic Sphincter.*—This is by far the commonest site for intussusception, and any explanation of this condition must explain this distribution. Leichtenstern has emphasized the similarities of the ileo-colic sphincter to the anus, and Arthur Keith and Elliott have directed attention to it lately. The last few inches of the ileum form a powerful detrusor, closed by the tonic ileo-colic sphincter. As spasm and tenesmus at the anus produce first partial and then complete prolapse, so do they at the ilio-colic sphincter. In just the same way as piles above the sphincter ani lead to prolapse, so do the cluster of swollen Peyer's patches above the ileo-colic sphincter lead to the higher degrees of prolapse at that point. Since my attention was directed to this matter I have seen no specimen of ileo-cæcal intussusception in which more or less prolapse through the sphincter did not exist. But whereas the prolapsed tissues at the rectum project into the open air, at the ileo-colic sphincter they project into the ascending colon. Moreover, Professor Arthur Keith has pointed out that in mice the longitudinal bands of the colon apply the ileo-cæcal orifice to the ascending colon in the later stages of digestion. In a condition of spasm they would apply a prolapse firmly to the orifice of the ascending colon until it was grasped in a contraction. Considering how common prolapse of the anus is in children, it is really remarkable that intussusception is so comparatively rare.

(v.) *Intussusception due to a Swollen and Hypertrophied Peyer's Patch.*—Intussusceptions frequently originate some 3 to 6 inches above the ileo-cæcal valve in a lateral dimple. In three such specimens I have dissected the dimple was due to dragging upon a swollen Peyer's patch which was found at the apex of the intussusceptum.

(vi.) *Paralytic and Relaxed Conditions of the Colon which permit of Accidental Invagination.*—I have seen two intussusceptions which had lasted a month and six weeks respectively, and both of which had reached the anus, one protruding 4 inches from it. Both were easily reduced, when the colon formed huge inert voluminous folds. In these cases I cannot help believing that the relaxed condition of the bowel permitted an accidental invagination of the cæcum or ileo-colic sphincter, and that the indolent contraction of the colon increased this invagination without strangling it.

(vii.) *Intussusception due to a Gangrenous Segment of Intestine which acted as a Foreign Body.*—A woman aged forty-nine was admitted to the London Hospital with a strangulated umbilical hernia. This was reduced. She passed blood from her anus and became collapsed. Mr. James Sherren therefore opened her abdomen, and found the central part of her transverse colon gangrenous. Her condition did not permit of a resection, and her abdomen was closed. She rallied, but the vomiting continued for seven days. Diarrhœa set in, and continued for three weeks. Nine days after the operation she passed a tubular slough 18½ inches long, smooth on its inner side, and rough and greyish-green outside. Eleven days after operation she passed 3½ inches of colon, with the mesentery attached and the longitudinal bands visible. She then recovered, but a stenosis subsequently formed, for which ileo-colostomy was successfully performed. In this case a gangrenous piece of intestine acted as a foreign body and was invaginated. The returning layer underwent necrosis first, so that the slough was inverted. Finally stenosis followed, as is so often the case after spontaneous elimination of the intussusceptum.

The Method of Growth in an Intussusception.—In simple ileo-colic intussusception the ileo-cæcal valve stands fast, whilst more and more ileum is prolapsed through its orifice into the cæcum. The process is limited by the strain on the mesentery, and by the orifice becoming choked by the swollen ileum and its mesentery. This form of intussusception would be more correctly described as an extensive and complete prolapse of ileum into the colon. Growth takes place entirely at the expense of the entering layer. Very frequently a secondary ileo-cæcal invagination is superadded.

In all other forms of intussusception, growth occurs entirely by additions from the sheath to the middle layer. The apex remains a fixed point held up by the entering layer and its mesentery. The sheath, which is the only active layer, grasps the intussusceptum firmly at the neck, and then endeavours to force it down the intestine by drawing the sheath up over it and by downward waves of peristalsis which slide the returning layer down upon the fixed axis of the mesentery and throw it into numerous folds. As the returning layer is driven down, it draws more and more of the sheath over the collar into the returning layer, and this fresh addition to the intussusceptum is grasped and driven down and compressed into folds at the next paroxysm of peristalsis. Thus it comes to pass in most cases that the entering layer and sheath are straight, but that the returning layer is folded and creased. As John Hunter long ago pointed out, 12 inches of intestine will form an intussusception 3 inches long; 3 inches will form the entering layer, and 3 inches the ensheathing layer, and 6 inches will be folded up in the returning layer.

Thus, in the ordinary ileo-cæcal form of intussusception, the ileo-cæcal valve remains at the apex of the intussusceptum, and the colon is progressively folded up on the termination of the ileum as an axis, so that by the time the descending colon forms the sheath, the cæcum, ascending and transverse colons are packed away in the returning layer. The great mobility of the colon in infants will even permit the ileo-cæcal valve to hang from the anus, where it may be recognized with the orifice of the appendix.

Varieties of Intussusception.—Intussusceptions are divided into enteric, entero-colic, and colic, according to their location. The enteric may be further subdivided into duodenal, jejunal, and ilean. The colic invaginations are the result of the passage of the ascending into the transverse colon, of the transverse into the descending, of the descending into the sigmoid flexure, or of the latter into the rectum. A little more detail is necessary in the case of the entero-colic group. This group is subdivided into (1) ileo-cæcal, (2) ileo-colic, (3) cæcal, (4) iliaca-ileo colica, and (5) the very rare appendicular.

In ileo-cæcal intussusception the ileo-cæcal valve forms the apex of the intussusceptum. In the ileo-colic form a longer or shorter piece of the ileum is prolapsed through the valve. In cæcal intussusception the inverted cæcum forms the apex of the intussusceptum, and the ileo-cæcal valve is laterally placed on the intussusceptum some distance from the apex. In the form known as iliaca ileo-colica the invagination commences one or two inches above the ileo-cæcal valve—usually, I believe, in a swollen Peyer's patch. When the intussusceptum reaches the valve, it may either thrust it into the colon in front of it, producing a secondary ileo-cæcal intussusception, or it may entirely or partially prolapse through the valve, and then produce a like result.

The relative proportion and importance of these subvarieties of entero-colic intussusceptions have been much debated lately. The older writers, such as Treves, Sargent, and Eccles, regarded the ileo-cæcal as much more common. More recent writers, such as Corner, Wallace, and Fagge, state that true ileo-cæcal intussusception is less common than was formerly supposed.

This is largely a matter of words and degree. The term "ileo-cæcal" has been used in two senses—to denote (1) any (entero-colic) intussusception in the ileo-cæcal region, and (2) a special form in which the ileo-cæcal valve heads the intussusceptum. On the other hand, true ileo-cæcal intussusceptions can scarcely occur at all unless there is some degree of prolapse through the valve which can be grasped by the ascending colon. It is, then, a question of degree when this prolapse deserves the name of "ileo-colic." The older surgeons did not distinguish subvarieties, and the cases they recorded as "ileo-cæcal" vitiate all the older statistics, whilst some of the younger surgeons are inclined to regard even a slight prolapse as indicating an "ileo-colic" intussusception. Wallace's figures, though small, are reliable: 19 entero-colic intussusceptions included 10 single, of which 8 were ileo-cæcal and 2 were cæcal; and 9 double, of which 5 were ileo-colic and ileo-cæcal, and 4 were enteric and ileo-cæcal. Since 1900 the registrars of the London Hospital have distinguished the subvarieties of entero-colic intussusception in 92 cases. These were distributed as follows: Ileo-cæcal, 51; ileo-colic, 20; cæcal, 21.

Multiple Intussusceptions in the Living.—More than one inflamed adherent and obstructive intussusception may be found in one case, but this is a very rare occurrence. Mr. D'Arcy Power has recorded such a case in a male baby five months old. In addition to an ileo-cæcal invagination 2 inches long, there was a retrograde intussusception of the transverse into the ascending colon. Both were adherent and covered by lymph. Mr. Power has found two further cases recorded by Peregrine and Dr. Handfield Jones, and a further case has occurred at the London Hospital.

Invaginations of the Death-Agony.—When the abdomen of a child who has died of meningitis, peritonitis, or abdominal traumatism, is opened, a number of intussusceptions are frequently found in the small intestine. They are usually retrograde in character, very short, and are neither adherent nor inflamed, so that they are readily reduced. There is no evidence of obstruction above them, and they produce no symptoms. Indeed, they are produced by the tumultuous waves of peristalsis which may immediately precede death in such case. In a few instances they have been found in association with other causes of obstruction, such as a fæcal tumour in the sigmoid flexure or ancient adhesions from chronic peritonitis.

Ascending or Retrograde Intussusceptions.—Intussusceptions of the living are nearly always descending. Leichtenstern collected 593 cases, and of these only eight were ascending. He is dissatisfied even with these eight, for he states that in all peritonitis was present, and they occurred under conditions in which retroperistalsis was already established.

A retrograde intussusception, however, could scarcely originate in any other way than by a reversal of the peristaltic waves, and it is interesting that so many of the authentic cases have occurred in the colon in the region of normal antiperistalsis. Thus, Besnier has reported a case of a simple invagination of the sigmoid into the descending colon, which was adherent and of nine days' duration. D'Arcy Power has recorded one of the transverse into the ascending colon, and Jones (quoted by Nothnagel) an invagination of the descending into the transverse colon which survived eight weeks.

On the other hand, Hektoen had a case in which four ascending invaginations were found in the ileum, and my colleague, Mr. Hugh Rigby, encountered a gangrenous retrograde invagination in the ileum at the London Hospital in January, 1903.

If descending intussusceptions be due to the swallowing of one part of the gut by that below it, as D'Arcy Power has said, then the ascending form may be described as due to the vomiting of part of the bowel through that above it (see John Hunter on "Emetics and Retroperistalsis").

Double, Triple, Quadruple, and Compound Intussusceptions.—The layers of a simple intussusception may be so firmly fixed together that the entire mass acts as one foreign body, and is ensheathed by the gut below. In this case a double intussusception is produced, composed of five tubes or ten layers. The same process may be repeated twice or even thrice. The double and triple forms occur in the intussusceptions of the dying. It is also not uncommon when an inverted Meckel's diverticulum is the origin of the intussusception. Sir Watson Cheyne has reported a triple intussusception the cause of which was an inverted diverticulum with a congenital stricture below it. When an enteric intussusception reaches the ileo-cæcal valve, it may either be arrested or pass a certain distance through it. In either case the valve is often invaginated into the colon, and a secondary ileo-cæcal intussusception is superadded.

In some cases of double invagination it would appear that the ensheathing coat has simply become folded or wrinkled up upon the intussusceptum. The secondary intussusception is often then retrograde in direction. A good example of this condition has been figured by Mr. D'Arcy Power. Finally, it is said that the coil of gut above may become invaginated into the entering layer, and in this way produce a double intussusception. This would seem to be the place at which to mention those instances in which other coils of gut or omentum have been caught between the neck and collar of an intussusception, and drawn into the gap between the entering and returning layers. Mr. D'Arcy Power exhibited such a specimen at the Pathological Society, for Mr. Lucy of Plymouth. In this case 3 feet of upper ileum was strangled between the entering and returning layers of an iliac intussusception lower down the intestine. My colleague, Mr. Hugh Rigby, has encountered a similar case where the lower angle of the duodenum was caught in a like manner, but was not strangulated, and Dr. Delépine has reported an almost identical case.

The Frequency of Intussusceptions with Regard to Incidence, Age, Sex, and Location.—Of all causes of intestinal obstruction, invaginations are by far the most common, and this is especially so in children. Out of a total of 669 cases of obstruction at the London Hospital from 1893 to 1905, no less than 189 were due to intussusception, a proportion of about 28 per cent. According to Leichtenstern, half of the 473 cases he collected were less than 10 years old, and one-fourth were less than 1 year old. Consecutive series give even a higher proportion. Thus, Eccles, from an analysis of the St. Bartholomew's Hospital registers, found that 27 of 40 cases were under 12 months of age (68 per cent.), and 37 of 40 cases under 10 years old (92 per cent.).

The following is the distribution according to age of 187 consecutive cases of intussusception at the London Hospital. These figures agree very nearly with those obtained from St. Bartholomew's Hospital, and are probably much nearer the truth than series of published cases such as Leichtenstern has collected:

Age.	No. of Cases.
0–1	135
1–10	30
10–20	3
20–30	4
30–40	4
40–50	6
50–60	2
60–70	2
70–80	1
Total	187

That is to say, 72 per cent. were less than 1 year old, and no less than 88 per cent. were under 10. The residue of 12 per cent. were fairly evenly distributed through the remaining years of life, during which period it will be seen that intussusception is a rare cause of obstruction. No more than 4 of 135 babies were less than 3 months old.

Intussusception is more common in males. The proportion at the London Hospital was 131 males to 58 males—that is to say, about 70 per cent. were males. Intussusception at and about the ileo-cæcal valve is far more frequent in children (70 per cent., Leichtenstern), but in adults enteric invaginations are nearly as common. Leichtenstern's figures for all ages are as follows: Enteric, 30 per cent.; entero-colic, 52 per cent.; colonic, 18 per cent. Once again Leichtenstern's deductions are vitiated by the source of his figures. The London Hospital records of 110 consecutive cases of all ages show that enteric and colic invaginations are much rarer. These cases were distributed as follows: Enteric, 10 cases = 9 per cent.; entero-colic, 92 cases = 84 per cent.; colic, 8 cases = 7 per cent. Of the entero-colic cases 1 was due to carcinoma of the ileo-cæcal valve. Of the colic invaginations 1 arose from a myxomatous tumour, and 1 from a carcinoma. Of the enteric intussusceptions, 2 originated in myomatous polypi, 1 in a carcinoma, 1 in an inverted Meckel's diverticulum, and 1 was retrograde. Enteric intussusceptions occur most often in the lower ileum. Colonic invaginations are commoner in the descending colon and sigmoid flexure.

Pathological Changes in an Intussusception.—*The mesentery and mesocolon*, which are drawn in between the entering and returning layers of an intussusception, are subjected to tension, and very frequently to torsion and strangulation. It has already been explained that the mesentery of the entering layer forms an axis upon which the returning and ensheathing layers are piled up. The tension on this axis necessarily becomes greater and greater, so that the intussusception becomes curved towards the mesentery. This curvature is greatest in the entering layer, and a deep crease is often produced upon its concave side. The drag of this taut axis upon one lip of the orifice of the intussusceptum reduces this to a vertical slit, directed eccentrically against the mesenterial wall of the sheath. These tension effects are only constantly seen in ileo-cæcal intussusceptions. They are not always found in colonic and enteric intussusceptions, and rectal invaginations are stated to be always straight.

In advanced entero-colic intussusceptions, in which the valve hangs from the anus, several curious points arise in connection with the mesentery and mesocolon. In the first place it is difficult to understand how the short piece of mesentery to the last three or four inches of the ileum can permit the ileo-cæcal valve to traverse the colon from end to end, and even project from the anus. It must, however, be remembered that this portion of mesentery is attached about the promontory at the centre of the abdomen. From this centre the mesentery swings round like the hand of a clock, and since the length of the entering layer is not increased, no extra length of mesentery is required to reach the intussusception as it travels along the colon around this centre. Very little attention has been paid to the changes in the mesocolon which contribute to this curious phenomenon. The mesocolon in infants is extraordinarily loose and long. This permits each part of the colon in turn to be drawn towards the promontory, around which the intussusception travels in quite a small circle until it passes vertically down the rectum and out of the anus, following a course like ʔ or a shepherd's hook. When the process is complete, the aperture within the hook is not larger than a shilling, and leads into a large hernial sac, which contains the upper part of the small intestine. The ileum issues through the hook to enter the intussusception. The anterior boundary of this hernial sac is formed by the mesenteries of the ascending, transverse and descending colons and the sigmoid flexure, which have been drawn inwards towards the ring around the promontory. The final arrangement is not unlike a Japanese fan, in which the handle represents the mesentery of the ileum, and the periphery is attached around the circumference of the abdomen, whilst the entrance to the hernial sac is to the right of the handle where it joins the fan.

Moreover, it is obvious that as the ileo-cæcal valve travels round the circumference of the circle it must produce one complete twist of the mesentery of the ileum, which is fixed at its other end to the promontory of the sacrum. Indeed, the ileo-cæcal valve is sometimes twisted more than once, and it then communicates these torsions to the mesentery, mesocolon, and to the intussusceptum.

The great cause of the obstruction and strangulation of an intussusception is the active and vital contraction of the sheath, and especially at its neck. It must never be forgotten that the sheath regards the intussusceptum as a foreign body which is violently stimulating its lining mucous membrane. The sheath, therefore, always is endeavouring to establish a ring contraction above the intussusceptum, and to squeeze it down the intestine. John Hunter pointed this out long ago, deducing it from the deep impression made by the appendix on the entering layer. Moreover, in that chronic form of intussusception in which the colon is voluminous, languid, and paralytic, neither obstruction nor strangulation occur. In such a case I have seen a child pass a motion from an ileo-cæcal valve hanging from the anus, and I have afterwards reduced the intussusception, which had existed six weeks, without the slightest difficulty. Other causes of obstruction which have been given are the curving, kinking, and torsion, of the intussusceptnm, and the great swelling which results from strangulation. Moreover, the orifice is slit-like and applied to the side of the sheath.

An acute intussusception rapidly tends to become irreducible. Thus, Gibson has stated that on the first day 94 per cent. are reducible, on the second 83 per cent., and on the third day only 61 per cent. The question of reducibility is of paramount importance, for I can find no record of a case in which an intussusception has been successfully resected in a child under a year old, although the point has many times been publicly challenged.

The chief cause of irreducibility is the swelling, stiffening, and thickening, of the layers of the intussusceptum due to œdema and extravasation of blood, the results of strangulation. These changes are best marked at the apex of the intussusceptum, and it is therefore usually the last few inches which are irreducible.

Reduction is very often impossible when an enteric or ileocolic intussusception has passed through the ileo-cæcal valve and is tightly strangled there. In subacute and chronic cases, adhesions between the entering and returning layers may prevent reduction, but most chronic intussusceptions are neither strangled nor inflamed, and therefore are not adherent. Torsion of the intussusceptum is a rare cause of irreducibility.

In acute intussusception, *the gut above the obstruction* is little altered, except that it is dilated and congested if the case is of more than two days' duration. In chronic cases it may undergo hypertrophy, and those other changes which have been described under Chronic Stenosis. Very rarely perforation has occurred above the intussusception.

The intussuscipiens seldom shows gross changes. In chronic cases it is sometimes thickened, and in acute cases which have terminated fatally it is not uncommon to find patches of ulceration and gangrene opposite the point where the apex of the intussusceptum presses against it. The intussusceptum may actually project through a rent in the sheath into a peritoneal cavity.

The intussuscipiens is sometimes creased and folded, and when one of these folds is very large, a double intussusception is produced. If the fold project upward beneath the upper part of the sheath, the secondary intussusception is, as in D'Arcy Power's case, retrograde. If the lower part of the sheath is folded outside the upper, then the secondary intussusception is descending.

The pathological changes in the intussusceptum are the most important in an invagination. The veins of the mesentery and mesocolon are compressed by the sheath, especially at the neck, in acute cases, and as a result the intussusceptum becomes engorged, œdematous, swollen, and finally gangrenous. The microscopic changes are those of œdema and profuse extravasation of blood into the cellular tissue from the ruptured capillaries, as in all strangled tissues.

The swelling is most marked at the apex and along the convexity of the intussusceptum, apparently because these parts are free from pressure and are most peripheral. The middle cylinder is as a rule more damaged than the central one, which is usually narrow and compressed.

In acute cases gangrene may appear as early as the third day, and those cases which survive pass the sloughs from ten days to three weeks from the acute onset.

The Spontaneous Elimination of the Intussusceptum.—The gangrenous bowel may be passed in small fragments, or in extensive and complete segments of the intestine exhibiting clearly such natural features as the mesentery, the longitudinal bands, the appendices epiploicæ, the vermiform appendix, or a Meckel's diverticulum. Cruveilhier has described such a specimen 3 metres long, and my colleague, Mr. James Sherren, has reported a case from the London Hospital in which two sloughs were passed 18½ inches and 3½ inches long respectively. The larger was inverted. They were portions of the transverse colon. When the inner and middle cylinders are adherent, the slough is passed with the same relation of parts as in the intussusceptum. When the middle layer separates first, the portion of gut has its peritoneum outside, as is normal; but when the entering layer gives way first, the bowel becomes inverted and is passed with its mucous membrane outside (Treves).

In an acutely strangulated intussusception, the gangrenous process is most advanced at the neck, where the collar cuts into it, and this is why large sloughs separate. In a chronic intussusception, on the other hand, the process is rather one of ulceration, and is located at the apex of the intussusceptum, from which it spreads upwards.

According to Leichtenstern, 40 per cent. of cases of spontaneous elimination do not recover. In some extravasation occurs around the neck, because the adhesions are incomplete. In others the separated intussusceptum obstructs the bowel lower down, and the adhesions give way. Some die of hæmorrhage during separation. In others ulceration follows, and carries the patient off by diarrhœa and exhaustion, or more swiftly by perforation. Occasionally the stump of the intussusceptum has led to a fresh and fatal intussusception, or a stricture has formed at the line of separation, as occurred in Mr. Sherren's case already referred to.

Leichtenstern in his monograph on intussusception has worked out all the statistics of this interesting subject. He states that the incidence is 42 per cent. of his series of recorded cases of intussusception. Spontaneous elimination has occurred once in 68 consecutive cases at St. Bartholomew's Hospital, and once in 189 consecutive cases at the London Hospital. The true incidence is therefore less than 1 per cent. Leichtenstern states that spontaneous elimination occurs in 61 per cent. of enteric invaginations, in 28 per cent. of colic intussusceptions, and in only 20 per cent. of ileo-cæcal invaginations. It is very rare in babies, and uncommon before six years of age, from which time onwards about 40 per cent. of intussusceptions undergo spontaneous elimination. Most of the sloughs are passed from seven days to four weeks after the onset. When the slough is separating, the motions become extremely foul and stained with blood. In some cases profuse diarrhœa heralds the appearance of the slough, in others constipation.

The older the patient, the less likely is he to recover after spontaneous elimination.

Sepsis and Infection.—Those changes which have already been described under the general heading of Strangulated Intestine occur in an intussusception. After about two days or less, micro-organisms pass into the tissues of the invagination, and even through them to the peritoneum. Inflammation, peritonitis, sapræmia, and even septicæmia, are the results, so that even when an intussusception has been successfully reduced, a number of patients cannot recover because of the advanced stage to which these infective processes have already attained.

Where a carcinoma of the intestine has produced a chronic invagination, it tends to infiltrate the returning rather than the entering layer. In a specimen of this sort in the museum of the London Hospital, which was obtained from a patient of Mr. F. S. Eve's, the returning layer was almost entirely formed of an inverted cylinder of new growth which had originated in the ileo-cæcal valve.

The Symptoms of Intussusception.—Nearly three-quarters of the cases of intussusception occur during the first year of life. These cases form a well-marked group, presenting regular and typical symptoms and a clear clinical entity. The remaining quarter of the cases are distributed over the rest of life. They occur unexpectedly, infrequently, and are most irregular in their symptoms, so that it is impossible to draw a common clinical picture of them. It is therefore advisable to first describe the definite clinical picture presented by intussusception in babies, and then to describe the chronic form which is more common in older cases.

Acute Intussusception in Babies.—A baby who has previously been in good health, and is often a very fine child (Hunter), and more frequently a boy, is suddenly seized with the most violent intestinal colic. The tiny sufferer draws up its knees as the attacks come on, and screams lustily, whilst its face appears to exhibit pain and astonishment. Soon after the onset of the pain the child vomits once or twice, but the vomiting is not urgent and may soon remit. Another early symptom is tenesmus. The baby strains almost incessantly, and passes small motions of blood and slime at frequent intervals. When the abdomen is examined, a tumour will be found in 75 per cent. of the cases if an anæsthetic is given. The tumour is most frequently found in the left loin, but may be felt at any point in the colon where it is accessible to palpation. The tumour is sausage-shaped, and about an inch in diameter and of variable length ; it hardens and becomes more definite during the spasms of pain, but may nearly disappear in the intervals. The abdomen is not distended during the first two or three days, and when this sign does appear it is of bad prognosis. The degree of rigidity and tenderness of the abdominal wall are difficult to define. As a rule the child resists attempts to handle the tumour, and these appear to excite attacks of spasm ; but as the little sufferer becomes more and more exhausted it resists manipulation less and less. In about a quarter of the cases the apex of the intussusceptum may be felt from the rectum. It is said to resemble the os uteri. It may even project from the anus, but this is not so common as in chronic cases.

The progress is from bad to worse. The attacks of pain work up to a climax after some hours, when the pain is continuous and the attacks occur as exacerbations. The tenesmus becomes more and more frequent; the vomiting is variable. Soon the child becomes quiet, collapsed, and ringed around the eyes; after two days distension begins, the temperature frequently rises, and the rigidity and tenderness of peritonitis appear. If unrelieved, the child dies from the second to the seventh day. Mr. McAdam Eccles's analysis of the symptoms in 40 consecutive cases of intussusception at St. Bartholomew's Hospital is most valuable. Twenty-six out of the 40 cases showed abdominal pain, vomiting, and the passage of blood and mucus. Thirty-four out of the 40 cases passed blood and mucus, and a similar number vomited. In 32 out of the 40 cases a tumour was found, and in 10 it was felt by rectum. Pain was absent in 5 cases. Blood and mucus were not observed in 3 cases. The above description applies to practically all cases under one year old. They are almost always acute and of the entero-colic variety.

The Differential Diagnosis between Acute Intussusception and Colitis in Babies.—The only condition which closely resembles acute intussusception in babies is acute colitis. This resemblance is not surprising, for the colon is acutely inflamed in intussusception, but in addition the intestine is completely obstructed. These two conditions are alike in the following particulars: They both attack babies, and usually before they are one year old. They both are frequently preceded by slight alimentary disturbance, such as vomiting and diarrhœa. Blood and mucus are passed with tenesmus in each disease. There is nothing to choose between the collapse in either case. Much has been made of the presence of pyrexia in colitis by some writers, but cases of intussusception frequently have pyrexia early in the attack. Even the presence of or absence of a sausage-shaped tumour may fail the physician, for it is admittedly not capable of palpation in a quarter of the cases of intussusception, and the sigmoid flexure in spasm may be palpated in the left iliac fossa of many cases of colitis. This tumour is, however, no thicker than the little finger, and does not so obviously harden and soften as in the case of an intussusception.

The crucial point in the diagnosis is whether complete intestinal obstruction is present or not. This is ascertained by searching for bile mixed with the blood and mucus on the napkin of the baby. If it is present after the first motion or two, the case is one of colitis, or a chronic intussusception. It it is absent, the baby is suffering from acute intussusception. The two Sisters of our children's wards at the London Hospital have observed this sign in 48 cases. In 43 cases of intussusception, there was no bile on the napkin in 36. It was present in 5 cases; of these 4 were chronic intussusception, and in 1 case the sign failed. In 2 cases the bowels were not open after admission to the hospital. In 5 cases of acute colitis admitted as intussusceptions, bile was present in 4, and was not observed in 1 case; but at the autopsy on this case bile was found all along the colon. The second important point in diagnosis is the character of the onset. In acute intussusception this is almost always absolutely sudden. In acute colitis the onset is as a rule more gradual, and is led up to by some vomiting and diarrhœa, but, unfortunately, a few cases of intussusception complicate gastro-enteritis.

Those cases of intussusception which occur after the first year are very variable in their symptoms. They may, however, be divided into acute or subacute cases, chronic cases, and chronic cases which terminate acutely. The acute cases do not differ very greatly from the above description, with due allowances for the age, and with the reservation that in older children and adults the symptoms tend to be subacute.

Cases of chronic intussusception form a fairly well-marked group.

The Symptoms of Chronic Intussusception.—These cases are often obscure and misleading. Rafinesque collected 55 cases, and of these no less than 27 were incorrectly diagnosed. Chronic intussusception has been mistaken for chronic colitis, cancer of the colon, fæcal accumulation, tubercular peritonitis, dyspepsia, gastro-enteritis, floating spleen, polypus and prolapse of the rectum, and many other conditions. There is no symptom which may not be absent or modified. A careful review, however, of each case will as a rule supply enough on which to found a probable diagnosis. The duration of chronic intussusception is from a month to a year or more. The onset is acute in about 30 per cent. of the cases; the symptoms then are alleviated, and the case runs a chronic course. In the majority of cases the onset is obscure and progressive, commencing with transient attacks of colic and irregularity of the bowels.

The feature of nearly all cases is abdominal attacks, which occur at irregular intervals, and tend to be brought on by the taking of food and aperients. These attacks occur at shorter intervals, and are more severe as the case progresses. They are of the nature of colic. The bowels are very frequently altered during the attacks. If the intussusception is entero-colic or colic, mucus is passed at frequent intervals during the attacks, with a little blood. When the invagination is enteric the bowels are more often constipated. Should a tumour be present in the abdomen, as it is in about half the cases, it becomes harder, larger, and more obvious, when spasm is present and moves along the colon. Thus, in a case on which the writer operated, and which was due to a myxomatous tumour in the left half of the transverse colon, it was only during the attacks that the tumour could be felt in the left loin. When the attack subsided, the tumour retreated beneath the left costal margin. When the pain is severe, vomiting is frequently present, and this is more likely to be the case if the invagination is enteric or entero-colic. Chronic intussusception is one form of stenosis of the bowel, and as a consequence dilated and contracting coils of intestine are seen in the abdomen in most cases, and especially during attacks.

The subject of a chronic invagination emaciates and becomes anæmic as a result of the incessant pain, diarrhœa, and vomiting, and because the taking of food often precipitates an attack, and is therefore avoided.

Chronic intussusceptions terminate by becoming acute, by perforation, or by reaching the rectum and anus. When they become acute, profuse vomiting and absolute constipation appear, and the patient often sinks in an hour or two.

I have encountered two cases which terminated in this way. In both the signs and diagnosis for months had been new growth of the right lung and pleura. Both patients began to complain of abdominal pain and uneasiness, to which little attention was paid. In each case the onset of total obstruction was ultra-acute —one case was dead in five hours. The condition found at the autopsies was alike in both. A more or less pedunculated secondary growth of sarcoma had formed in the ileum, and had produced invagination. Curiously enough, a free Meckel's diverticulum was present in both cases.

Perforation occurred 12 times in 35 cases of chronic intussusception collected by Rafinesque; 1 was some distance above the intussusception, 3 were just above it. In 15 cases the sheath was perforated with hernia of the intussusception, in 3 cases without. In 1 case the sheath was torn completely across.

The intussusceptum reaches the rectum in nearly one-third of the cases (Rafinesque, 16 of 55 cases). Even when it has protruded some inches from the anus, motions may be passed from the ileo-cæcal valve. The sphincter is often patulous and relaxed.

The Prognosis in Intussusception—*Spontaneous Cure.*—The chance of spontaneous cure does not amount to more than 1 or 2 per cent. This may take place either by (1) spontaneous reduction, a phenomenon several times recorded, but always dubious; (2) by spontaneous elimination of the intussusceptum; or (3) by a fæcal fistula forming above the invagination.

Spontaneous Reduction may have occurred in a child admitted, under the writer, at the London Hospital. Three times a medical man reduced a definite abdominal tumour by injections of warm water in a child suffering from the symptoms of intussusception. When the tumour reappeared a fourth time, the child was sent into hospital. On admission no tumour was present, and none reappeared.

In a chronic invagination of the transverse into the descending colon due to a myxomatous tumour, when I opened the abdomen and drew out the gut the intussusception was reduced, although the bowel showed plenty of signs of invagination. The long history of this case, with intervals of perfect recovery, led one to suppose that complete spontaneous reduction had often occurred before. Nothnagel records a similar case. It must be remembered that the upper part of the colon is the region of retroperistalsis, and therefore spontaneous reduction is theoretically not unlikely.

Spontaneous Elimination of the intussusceptum with recovery is a very rare occurrence. It has occurred once in 189 cases at the London Hospital, and once in 68 cases at St. Bartholomew's Hospital. The fallacy of Leichtenstern's collected series is shown by the fact that he found that in 42 per cent. of all his cases spontaneous elimination occurred; 40 per cent. of these died subsequently. When treated under favourable conditions, about 50 per cent. of the cases recover. At the London Hospital, from 1893 to 1895, 55 cases were treated, with a mortality of 82 per cent.; but from 1900 to 1905 the mortality fell to 57 per cent. on 134 cases. Mr. F. S. Eve has published a consecutive series of

11 cases submitted to laparotomy, with 43·3 per cent. mortality. Mr. C. H. Fagge has published 18 consecutive cases, with 50 per cent. mortality. The splendid results that may be obtained in a favourable series of cases by a skilled operator are shown by Mr. C. Wallace's series of 20 cases, with 4 deaths. Of these, only 2 were irreducible, and only 4 of more than forty-eight hours' duration. The prognosis in chronic cases is much more favourable. I have operated on only 4 such cases, and all recovered.

Irreducibility is the fundamental point in the prognosis of intussusception in babies after treatment, for no successful cases of resection have been recorded under one year old. Gibson's figures may here be repeated: In the first day 94 per cent. of intussusceptions are reducible, during the second 83 per cent., and during the third only 61 per cent.

When the child becomes quiet and the abdomen distended and rigid, the case is wellnigh hopeless.

The Treatment of Intussusception.—1. *In Babies*, the best results are obtained by rapid laparotomy and reduction, when an expert abdominal surgeon is immediately available and the surroundings are favourable. Where this is not the case, the best treatment is by injection of warm water or milk. It is best introduced by a rectal tube and funnel, with a drop of not more than 3 feet. The escape of fluid around the tube may be prevented by cotton-wool and vaseline and by holding the buttocks together. The child should be anæsthetized and the buttocks well raised. The tumour should be manipulated to assist reduction. The process may have to be repeated twice, or even thrice, before it is successful. The child should then be placed under morphia and belladonna to prevent recurrence, which is not uncommon (Eccles, of 23 cases treated by injection, 14 required subsequent laparotomy).

2. *In Adults*, intussusception is seldom so urgent a matter as in babies, and there is usually time to obtain a surgeon, as the treatment is entirely surgical.

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5. INTESTINAL OBSTRUCTION DUE TO VOLVULUS, INTERTWINING, AND KNOTTING OF INTESTINES.

Definition.—A *volvulus* of the intestine is said to be present when the bowel is twisted or has undergone axial rotation. Twisting may occur about three axes: (1) A piece of intestine may be twisted around its own longitudinal axis. (2) The common form of volvulus is when a coil of intestine rotates around its mesenteric axis, but twisting of the intestine around its own axis may also be present at the neck of the volvulus. (3) Finally, two coils of intestine possessing long mesenteries may twist or intertwine around one another, or even become knotted together. As a rule one of them forms an axis around which the other twists, as honeysuckle twines around a stick. The coil which forms the axis is that which is more severely strangled.

Incidence.—Twenty-seven cases of volvulus have occurred at the London Hospital in thirteen years among 669 cases of intestinal obstruction (1 in 25).

Etiology.—The mesenteries of the intestine are most beautifully constructed to permit of free movement, but to render volvulus impossible. The mesentery of the small intestines is particularly wonderful in its curves. These are so formed that every piece of gut has as its base of attachment the whole extent of the origin of the mesentery. It is only when this structure is altered that volvulus becomes possible. The causes which predispose to volvulus act in three ways:

1. They narrow the base of attachment of a coil of intestine and produce a pedicle.
2. They lengthen the mesentery of the coil.
3. They lead to an adhesion at the centre of the loop, which adhesion with the base of attachment forms an axis around which the loop readily rotates.

The base of attachment is narrowed in many ways; adhesions may form between coils of intestine, producing a fixed loop; or a gland in the mesentery may become inflamed, and in the process of resolution contract the mesentery of a coil of gut to a narrow pedicle. Coils of gut which have been strangled by a hernial ring or a band may retain their constricted neck, and subsequently rotate around it. In such a case I had divided a band encircling a coil of ileum and freed the strangled loop. The boy recovered, but some months later was readmitted under Mr. Hugh Rigby, who found a volvulus of the ileum around the constricted pedicle formed by the band. A chronic condition of inflammation may be set up and maintained in the mesentery by any long-continued source of irritation in the bowel, such as tubercular or stercoral ulceration. This is exceedingly common in the mesosigmoid, as was long ago pointed out by Treitz. This chronic inflammation produces stiffness and contraction of the mesentery, which in many cases leads to narrowing of the base of attachment.

An infant may be born with an unusually long mesentery to any part of the intestine, and this is especially frequent at the entero-colic junction when the cæcum and ascending coil fail to adhere to the right loin. The whole of the small intestine, cæcum, and ascending colon, may then have a common mesentery. More often lengthening of the mesentery is acquired, as when intestine frequently descends into a hernial sac, or the coil of gut is often distended or overloaded so that it falls into the pelvis. In other cases the elongation of the mesentery appears to be due to loss of fat and relaxation of the abdominal wall, the result of repeated child-bearing or advancing years.

I have collected three cases where an adhesion of the loop of intestine formed with the base of attachment an axis around which the coil rotated. In one the centre of the sigmoid flexure was adherent to the mesentery of the small intestine, and the flexure rotated on this axis. An inflamed coil of small intestine released from a band or hernial sac may become attached at its centre, and subsequently rotate in a similar manner around its lengthened mesentery and contracted base.

The Exciting Causes of Volvulus.—When the above-mentioned changes have taken place, so that a coil of intestine is capable of rotation, the final twist is produced either by some violent effort, such as lifting or straining, or by distension of the loop, when, as it finds room for itself in the abdominal cavity, it may undergo rotation. In a case of Mr. Jonathan Hutchinson, junior, in the London Hospital, a volvulus of the cæcum was apparently induced by the rupture of a pancreatic cyst which occurred in the hospital. The volvulus was treated successfully. The cyst then re-formed and was drained. The patient completely recovered.

When a coil of intestine has become rotated, the chief hindrance to its reduction is its distension. If this can be relieved, it may even spontaneously uncoil.

Other conditions which prevent reduction of the twist are the great size and weight of the coil, or another coil may be drawn across the twisted pedicle and compress it (Leichtenstern).

Pathology.—Many degrees of twisting occur. They vary not only in extent, from half a complete circle to even three or four turns, but also in their tightness.

When the twist is slack and the vessels are not constricted, the symptoms are those of partial obstruction. The pain is colicky, flatus and even solid fæces are passed, and the distension is not very great. When, however, the twist is tight and the veins are severely obstructed, the usual results of torsion of an organ result. Intense venous engorgement is produced, the bowel becomes dark purple and œdematous, and ultimately green or grey and gangrenous. The capillaries burst, and blood is extravasated into the tissues. The mucous membrane pours a profuse blood-stained exudate into the twisted coil, and the serous covering sweats sanguineous serum into the peritoneal cavity. The amount of this secretion and the proportion of blood in it vary with the severity of the strangulation of vessels. The enormous distension which so rapidly occurs in a twisted coil of gut is, however, almost entirely due to gas formation. The origin of this gas is a most interesting problem, to which we have already referred. The rapidity with which it forms would appear to exclude its origin from gas-forming bacteria unrestrained by the damaged gut. Probably the gas is carbonic acid produced by the vital processes of the intestine, which is not removed owing to the venous obstruction. The consequences of this distension are very marked when the coil is large, as in the case of the sigmoid flexure. The coil itself in the case of the colon may reach some 5 inches in diameter; it is greatly increased in length, and its walls are so stretched that the longitudinal striæ disappear. The peritoneum may actually split and patches of gangrene appear, but gross perforation very rarely occurs (Treves) in the loop itself. When perforation is present, it is above the twist. Within a day or two, however, transudation of micro-organisms occurs from the lumen of the coil to the peritoneum, and well-marked peritonitis is produced.

When the sigmoid is twisted, the huge coil extends up the abdominal cavity in front, forcing the small intestine backwards to the right, and preventing any marked distension of the intestines above the volvulus. The abdomen becomes extraordinarily tense. The diaphragm is forced up to the level of the fourth or even the third intercostal space, and the bases of the lungs and heart are compressed, so that circulation and respiration are seriously impeded.

Anatomy.—Volvulus is most common in the sigmoid flexure. It also occurs at the entero-colic junction and in the small intestine. Twenty-four cases of volvulus at the London Hospital were distributed as follows: Sigmoid flexure, 12; entero-colic, 5; small intestine, 7. Leichtenstern's figures are as follows: 76 cases—sigmoid flexure, 45; a loop of ileum, 23; the jejunum and ileum combined, 8. Curiously enough, he gives no cases of entero-colic volvulus.

1. *Volvulus of the Sigmoid Flexure.*—The sigmoid flexure may (*a*) twist about its mesenterial axis, or may (*b*) intertwine with a coil of small intestine.

(*a*) *The sigmoid flexure is twisted about its mesenterial axis.* This is the common form of volvulus. The determining causes appear to be chronic constipation and mesosigmoiditis. The loaded coil of intestine falls into the pelvis, and drags on its mesentery, so that this is not only lengthened, but its base of attachment is narrowed. The chronic inflammation in the mesosigmoid contracts its base and stiffens it. The flexure may rotate either forwards or backward. When it rotates forward the rectum lies behind the descending colon ("rectum en arrière," Potain). This is more common. When it rotates backward the rectum lies in front ("rectum en avant," Potain).

In consequence of its size, the changes in the sigmoid flexure when it is twisted are greater in degree than in volvulus elsewhere. For instance, the distension may be so great that the diaphragm is forced up to the level of the third rib.

(*b*) *The sigmoid flexure is intertwined with a coil of small intestine.* Leichtenstern collected twenty-one such cases, of whom all were males except one. Their ages varied from twenty-four to seventy-three. The condition is, however, extremely rare, and no case has been recorded at the London Hospital. The loops concerned must be of considerable length. Leichtenstern states that the loop of small intestine varies from 4 to 21 inches, and the sigmoid from 12 to 40 inches. As a rule (twelve in twenty-one cases, Leichtenstern) the coil of small intestine lies in front and forms the axis. The sigmoid pedicle is wound round it from behind forward. The sigmoid may, however, form the axis, or the small intestine may lie behind, so that four varieties have been described. Either or both coils may undergo rotation on their own axis in addition. It is not surprising that the clinical course of this condition is exceedingly violent. Many die on the day of the attack, and nearly all within two days. The symptoms are those of ultra-acute obstruction of the small intestine.

2. *Volvulus of the Entero-Colic Region.*—Three subvarieties of this form of volvulus have been described:

(1) Twisting of the normal ascending colon about its vertical axis.

(2) Twists limited to the cæcum.

(3) Volvulus of the termination of the ileum, cæcum, and ascending colon, due to the presence of a long mesentery to the ileo-colic junction.

I have carefully reviewed the cases (those of Curling, Fagge, and Handfield Jones) which are claimed to be examples of the first and second varieties of ileo-colic volvulus. They do not appear to me to warrant a belief in any such subdivisions.

The ileo-colic junction is frequently possessed of a long mesentery which is either a congenital deformity or an acquired condition. It will readily be understood that the pendulous loop so formed may undergo simple axial rotation in either direction around its mesenteric axis, or may intertwine with a loop of the small intestine. When rotation has occurred, the cæcum becomes enormously distended, and may travel from the right iliac fossa to almost any part of the abdomen. Should it pass to the right hypochondrium, the cæcum is turned over, and the ascending colon is doubled across transversely.

In a case of the late Dr. Schorstein's on which I operated, the mesentery of the cæcum was 11 inches long, and that of the ascending colon 9 inches. The cæcum had passed behind the whole mesentery of the small intestines to the region of the spleen, and had rotated twice on a pedicle formed by the ascending colon and ileum. This pedicle, as I have said, crossed behind the mesentery, and was constricted by it.

A similar condition in a man aged fifty-five was under the care of Mr. Mansell Moullin in 1905. At the operation the cæcum, the size of a melon, was found in the left hypochondrium. The notes state that " the pedicle, consisting of the ileum and ascending colon, was twisted one whole turn from left to right. Also another entire turn took the whole of the mesentery across the pedicle."

3. *Volvulus of the Small Intestine.*—This is of two varieties. Either one coil may twist about its mesenteric axis, or two loops may be intertwined. The latter is so rare an occurrence that it need not be further alluded to (see Leichtenstern). It has been already pointed out that a coil of small intestine surrounded by a band, a hernial ring, or the edges of an aperture, may undergo rotation on its axis; and this, indeed, may be the chief cause of obstruction. It must be clearly stated that normal small intestine and mesentery cannot undergo a pathological degree of torsion, and that some congenital or acquired deformity of the mesentery or some adhesion is necessary to render volvulus possible. It is necessary to state this, for where no other cause is found for intestinal obstruction or gangrene of small intestine, it is easy to assume that it was twisted. In this way gangrene from acute thrombosis of the portal vein has been regarded at the operation as due to volvulus.

It is probably not unusual for more or less twisting of the small intestine to take place without any obstruction either to the lumen or the vessels, but such a twist may conduce to an obstruction from some other cause. A patient of the writer's had passed a very large truncated gall-stone by the rectum after some days' partial obstruction. Some months later she was seized with acute and total obstruction, which was relieved by removing a smaller truncated stone from the upper jejunum at a point where the latter crossed the mesentery from left to right, and turned sharply round its edge. The volvulus, which apparently involved most of the small intestines, and was in no way fixed or strangulated, was left, and the patient made a good recovery. Mr. Mayo Robson has reported two cases, which he regards as volvulus, due to the passage of large gall-stones.

The ordinary form of volvulus of the small intestine illustrates what has been said of the general cases of volvulus—viz., that the mesentery is lengthened, the base of attachment narrowed, and the apex of the loop may be adherent.

A child may be born with its small intestine twisted; indeed, the distension so caused may obstruct labour. Dr. Drummond Maxwell has reported such a case, and placed the specimen in the London Hospital Museum. The volvulus involved some 2 feet of the ileum, which was black and gangrenous. Other cases have been described by Dr. N. Pitt and Mr. Harrison Cripps.

Symptoms of Volvulus.—It will be necessary to distinguish between volvulus (1) of the sigmoid flexure; (2) of the cæcal region; (3) of the small intestines.

1. *Volvulus of the Sigmoid Flexure.*—This is the common form of volvulus. The general type of symptoms in volvulus of the sigmoid flexure is that of acute intestinal obstruction, with certain characteristic signs, which make a diagnosis more or less certain and easy. The patients are nearly always men. The ratio of men to women is usually stated to be as 4 to 1. At the London Hospital no case of volvulus of the sigmoid flexure in a woman was recorded between 1893 and 1905, among twelve cases.

The onset is as a rule sudden, but seldom so sudden as in strangulation by band. *The pain* is severe, and exacerbations occur, which are often of the nature of colic. The characteristic point is the early appearance of *tenderness* due to peritonitis.

Vomiting is little marked, and in a case of my own was absent altogether. This is, perhaps, not surprising when we consider how low down the intestine the obstruction is placed. *Eructations* are, however, common.

Constipation is as a rule absolute, but considerable variations occur in this symptom, and *tenesmus* is frequently present.

Collapse is later in its appearance, and not nearly so well marked as in strangulation by band.

The respirations are often frequent, in consequence of the abdominal distension.

The abdominal condition is the characteristic sign of volvulus of the sigmoid flexure. The abdomen rapidly becomes distended and tense to an extraordinary degree. The diaphragm is forced upwards, and the respiration and circulation are correspondingly interfered with.

The distension is so great that, as a rule, no separate coil of gut can be distinguished. Great tenderness from peritonitis appears early. It is exceptional for contracting coils of intestine to be visible, and when this is so the case is subacute in its onset and course. Although volvulus of the sigmoid usually produces acute intestinal obstruction, yet in not a few cases the onset may be gradual and attended by colicky pains and the passage of flatus, or even fæces, in fair quantity. Diarrhœa may be present, and it is even probable that incomplete degrees of volvulus may either spontaneously untwist, or, if they remain twisted, may only partially obstruct the bowel and its vessels. In the latter case some passing atony of the muscular coat may produce complete obstruction. On the other hand, volvulus may pursue an ultra-acute course, as in a man aged thirty-eight, under the care of Mr. Warren Tay, in 1902, at the London Hospital, who seven hours after the onset of obstruction was profoundly collapsed, and his abdomen hard, resonant, and tightly distended. His sigmoid flexure was found to be black and gangrenous, and his peritoneum contained turbid blood-stained fluid.

2. *Volvulus of the Ileo-Colic Junction.*—Volvulus of the ileo-colic junction around an abnormal mesenterial axis is not a very rare condition. Five cases have occurred at the London Hospital, and the writer has personally examined three cases. Leichtenstern, who is usually so complete, appears to regard this form of volvulus as excessively rare, and treats the subject very briefly. The obstruction is usually subacute in character. The onset is even less sudden than in volvulus of the sigmoid flexure. Vomiting is present as a rule, but is not severe. The distension is not so great as in volvulus of the sigmoid, but in all four cases of which I have records the distended cæcum was located through the abdominal wall as a resonant tumour, the size of a child's head—twice in the left hypochondrium, and twice in the right loin and iliac fossa.

3. *Volvulus of the Small Intestine.*—There is nothing typical about the symptoms of this form of volvulus. They may be acute, subacute, or chronic, or attacks of obstruction may recur for a long period with spontaneous intervals of relief. In many cases the twisted coil can be felt as a tumour in the umbilical region, and vomiting is a marked and constant symptom.

Prognosis.—In the severe cases which come to operation, the prognosis is exceedingly bad. In thirteen years at the London Hospital, only two cases recovered out of twenty-seven (mortality, 93 per cent.). It is only fair to add that two further cases have been successfully operated on during 1906.

Treatment.—The treatment is surgical. The abdomen is opened. The distended coil is punctured, emptied, brought out, and the volvulus reduced. If it is gangrenous it is at once resected. Otherwise a Paul's tube is fixed in it, and, should the patient recover, the volvulus is resected subsequently, and end-to-end anastomosis performed. If the sigmoid flexure is simply emptied and reduced, the volvulus will frequently recur. Dr. Blake has recorded a very remarkable case which was successfully operated on four times for volvulus of the sigmoid flexure. Twice the intestine was incised and emptied, and twice a rectal tube was manipulated into the volvulus after the abdomen was opened.

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Torsion or Volvulus of the Great Omentum.—This condition was first described by Oberst in 1882. He found a pedunculated portion of omentum twisted in a hernial sac. In torsion of the omentum this structure is twisted upon a more or less narrow pedicle, so that the part peripheral to the twist becomes engorged with venous blood, or even gangrenous. Corner and Pinches in an elaborate paper collected 54 cases up to 1905. Of these, 35 cases were examples of *unipolar rotation*, in which the omentum had rotated on one pedicle. This was situated in the upper third of the omentum in 24 cases, in the middle in 1 case, and in the lower third in 10 cases. *Bipolar rotation* has been recorded in 6 cases; that is to say, the omentum was adherent at its extremity, and rotated on the axis formed by its two points of attachment. The secondary adhesion has been to a hernial sac, to the neck of a hernial sac, to the right Fallopian tube, and to an appendix epiploica or the ascending colon.

Intertwining of pedunculated omental tumours has been reported by Heitz and Bender, and by Mauclaire. In Eitel's patient, in whom the torsion was not tight and the course of the case was chronic, many gallons of ascitic fluid were drawn off by puncture.

Symptoms.—There is often a history of previous attacks, which are no doubt due in many cases to loose or reducible torsions of the omental mass.

The majority of the patients are men of advanced years who have suffered from reducible inguinal herniæ for years. The onset of the symptoms is frequently associated with some trouble with the hernia and forcible attempts at reduction. Where no hernia exists, or where the omentum is within the abdomen when it becomes twisted, the onset of the disease is sudden and without warning or introduction. The patient is seized with sudden and severe abdominal pain, which is general at first, but is as a rule later referred to the right and lower part of the abdomen. Nausea and vomiting are usually present, and are proportional to the pain.

A tumour is nearly always palpable unless the abdominal wall is too rigid or fat. Frequently it is in the hernial sac or just inside the hernial ring. In other cases it is in the iliac fossa, and extends a variable distance up into the abdomen. The tumour increases in size. It is ill-defined, tender, immovable, and dull on percussion. After a day or two the signs of free fluid appear in the abdomen, and should an empty hernial sac be present, this is full, soft, and fluctuating. The abdomen becomes distended, and is rigid, especially over the tumour. The temperature is as a rule slightly raised, owing to the extravasated blood. The pulse-rate is seldom over 100 per minute unless the hæmorrhage be excessive. The bowels are constipated, but as a rule flatus is passed and enemas obtain a tardy result.

Pathology.—Where many twists are present, it is often clear that some have existed for a considerable time. These preliminary twists are probably responsible for previous attacks of strangulation, which pass off as the circulation adapts itself.

For torsion of the omentum to occur, a preliminary moulding of this structure is necessary. In twenty-eight of thirty-three cases collected by Stewart in 1905, these preparatory changes had taken place in a hernial sac. When the omentum habitually descends into a hernia, its extremity becomes a rounded swollen mass with a neck or pedicle, and very frequently one or more adhesions to the fundus of the sac or to the hernial ring. These changes in form of the omentum are the predisposing causes of torsion. When these conditions are once produced, the pedunculated mass of omentum may be twisted either in the hernial sac or whilst it rests within the abdominal cavity. The exciting causes of volvulus are—(1) Reduction and redescent of the omental mass along the inguinal canal; (2) fortuitous external movements communicated to the mass, especially by the legs, in walking; (3) within the abdominal cavity unequal contraction of the abdominal wall, respiratory movements, and the peristalsis of the intestine, probably convey the rotatory impulse to an omental mass which is unsymmetrically hung upon its pedicle.

Where no hernia has been present, the omentum has for the most part been found adherent at its extremity. In Noble's case it was adherent to the right Fallopian tube; in Wiener's case to an appendix epiploica on the ascending colon. The omentum, being attached at either end, is rolled around this axis (bipolar rotation). It is of course evident that torsion in such a case must occur at each end in opposite directions. Scudder has recorded a case in which no hernia was present, and the omentum was not adherent. The specimen is beautifully figured. The whole of the omentum may be twisted or only a part of its end, or two separate pedunculated portions may be intertwined.

The *pedicle* varies from ½ inch to 6 inches in length, and may be twisted from one to eight times.

The *Diagnosis* is from incarcerated hernia, reduction *en masse*, appendicitis, a twisted ovarian tumour, or intestinal obstruction, as in Audier's case. When Rudolf collected the first twenty-nine cases, only one had been diagnosed correctly.

The *Treatment* is surgical. A hernia, if present, is explored. An incision is then made on the tumour, and when the infiltrated omentum has been freed from adhesions, ligatures are applied above the twisted pedicle and the mass removed.

In Eitel's chronic ascitic case the omentum was unrolled and spread over the intestines. The patient was cured.

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A LECTURE ON GASTRIC SURGERY*

THE ANATOMY AND PHYSIOLOGY OF THE STOMACH

Gentlemen,—It is always a sound principle to commence the consideration of the diseases of any organ with a review of the anatomy and physiology of that organ, especially of the more recent advantages on these subjects. We shall therefore review to-day those parts of the development, anatomy and physiology of the stomach which seem most clearly to elucidate the nature and treatment of its diseases.

1. Development.

The stomach in its early stages of development is a median organ in which the anterior border to which the gastro-hepatic omentum is attached looks directly forward, whilst the posterior border and the gastro-splenic omentum are behind. It is the rapid growth of the great curvature and the budding out of the cardiac fundus that lead to the rotation of the organ, so that its posterior border passes to the left, and its anterior border, although to a much less degree, is displaced to the right. It will at once be seen that the left surface of the fœtal stomach thus becomes anterior, and the right posterior. The application of this fact to surgery is in connection with referred pains. There is some evidence, although perhaps not conclusive evidence, to show that the referred pain from an ulcer on the anterior wall of the stomach is more marked on the left side, and of one on the posterior wall on the right side, of the back and epigastrium.

As Professor Arthur Keith has pointed out, the cardiac fundus sprouts out from the stomach in a very similar way to that in which the cæcum buds out from the colon. This separate development appears to indicate a separate function for this part of the stomach, and the cardiac fundus certainly has a separate blood and lymphatic supply. The oxyntic or border cells are more numerous in the glands of the cardiac fundus, and the study of the movements of the stomach shows that the fundus is chiefly used as a reservoir from which the actively churning pyloric portion is fed. We shall see, when we come to discuss the diseases of the stomach, that ulcer is very rare on the cardiac fundus, perhaps because the food is not actively churned up in this part. The separate lymphatic supply of the cardia has an important bearing on cancer; for in removing a cancer of the pyloric part it is practically safe to leave the fundus, in which there will probably be no secondary deposits. Conversely, cancer of the fundus, when it does occur, which is seldom, does not tend to spread to the rest of the organ, and is peculiarly liable to lead to cancerous enlargement of the glands of the left posterior triangle of the neck.

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2. ANATOMY.

The position and axis of the stomach have been the subject of much misapprehension, apparently because the position of the stomach was studied after death, when the diaphragm was in a condition of complete expiration, and the abdominal wall was either completely relaxed or entirely removed, and the stomach itself was in a condition of paralytic dilatation. Observations on the living during operation, or by means of bismuth emulsion and the Röntgen rays, indicate that during life the stomach is nearly vertically placed. Its cardiac orifice is just to the left of the middle line, under the seventh left costal cartilage. The pylorus is just to the right, about a hand's-breadth lower down. The two orifices are only separated from one another laterally by the width of the sternum. Many people consider that the stomach is placed transversely across the abdomen; but this is, of course, an error, as the cardiac orifice is deeply placed just to the left of the spine, whilst the pylorus is nearer the anterior abdominal wall, so that the axis is obliquely placed and directed from behind and the left down forwards and to the right. The small curve and the orifices are, moreover, nearly fixed, whilst the fundus and great curve are capable of free movement, and it is by enlargement along these borders that increase of capacity within a wide range is provided for.

The Relations of the Stomach.—The organs which are in contact with the anterior and posterior surfaces of the stomach are those to which it will become adherent when ulcerated. In front the stomach is in contact with the liver, diaphragm, and abdominal wall. When an ulcer perforates the area in contact with the liver, it is frequently sealed off by adhesions between the two. This seldom occurs in the two areas in contact with the diaphragm and abdominal wall, presumably because these parts are in constant and active movements. The posterior relations of the stomach are chiefly of surgical interest, because large chronic ulcers frequently remove large areas of the stomach wall, and leave these organs exposed in the floor of the ulcer. The anterior wall of the stomach looks towards the greater sac of the peritoneum, and perforation there will involve that cavity; whilst the posterior wall is in the lesser sac of the peritoneum, and a perforation will first involve the lesser sac, and may not spread farther if the foramen of Winslow be sealed off. The second part of the duodenum is placed immediately over the hilum of the right kidney, to which it is attached. When the kidney slips downwards and rotates forward on the renal artery, the second part of the duodenum may be twisted and kinked so as to obstruct its lumen. The stomach will then become dilated. This association of dilated stomach with right movable kidney has long been recognized. A case of this sort was transferred to me at the London Hospital by Dr. Robert Hutchison. She was a woman of about thirty-five years of age, with typical dropping of the stomach. The lesser curve was clearly felt and seen below the liver margin, and the greater curve descended far below the umbilicus when she stood up. She had all the symptoms of dilated stomach—vomiting at long intervals of great quantities of fermenting food, and progressive wasting. The interesting point, however, was that when she was put to bed, and the foot of the bed raised, the stomach emptied itself, and she rapidly put on flesh. After this had happened once or twice, with relapses as soon as she stood up, Dr. Hutchison transferred her to me for gastro-plication.

On examining her, however, I found that the right kidney was excessively movable, and decided to fix that first, as this operation involved very little risk. The result was most satisfactory, and she was then able to stand up without kinking her duodenum and dilating her stomach.

The arteries in relation with the stomach are of surgical importance, not only because ulceration leads to hæmorrhage from them, but also because their position must be known in order that they may be dealt with in operations on the viscus.

The coronary, splenic and pancreatico-duodenal are those from which arterial hæmatemesis most frequently occurs. The coronary artery is not only in relation with that part of the stomach which is most frequently ulcerated, but it is in contrast with the gastro-epiploic arteries in very close contact with the stomach wall, no doubt because the lesser curve is a fixed point, and the greater curve advances and recedes between the layers of the greater omentum. These facts together account for the great frequency of hæmorrhage from this artery. The splenic artery is exposed by ulcers of the posterior wall, which lay bare the pancreas, whilst the pancreatico-duodenal artery is in contact with the posterior surface of the pyloric antrum—a very common site of ulcer.

The veins of the stomach present several points of surgical interest. Very rarely hæmatemesis, in cases of gastric ulcer, occurs from them, but they so readily thrombose in this condition that this seldom occurs.

In cirrhosis of the liver, however, when the obstructed portal vein is engorged with blood, the most profuse and rapidly fatal hæmorrhages may occur from very numerous points on the gastric mucous membrane. In some cases, however, no such point can be found from which the blood has issued, and then it may be explained in some cases by supposing that the hæmorrhage took place from the large collateral veins which pass through the cardiac sphincter into the œsophageal veins. In advanced cases of cirrhosis these veins are said to hang like bunches of distended piles into the stomach. When the patient has bled to death, the veins collapse and withdraw into the œsophagus, and the minute puncture from which the blood issued is overlooked.

The third point of interest about gastric veins is their possible association with post-operative hæmatemesis. In many of the recorded cases in which severe and even fatal hæmorrhage has followed an abdominal operation, the omentum has been ligatured or severely handled, and it is claimed by some that thrombosis spreads up its veins to the gastro-epiploic vessels, and leads to thrombosis in the gastric veins, and thence to hæmorrhage either from consequent congestion or digestion of the areas involved;

but this theory, though ingenious, lacks actual proof. In many instances the omentum has not been injured at all.

The lymphatic tissue in the mucous membrane of the stomach is aggregated into follicles, and is, according to most accounts, more abundant in the pyloric portion than in the body. This fact is perhaps connected with the greater frequency of ulceration there. According to Professor Arthur Keith, lymphoid tissue is found in larger quantities in the cardiac fundus.

Dr. Charles Miller was kind enough to show me sections from all parts of the stomach, the diseases of which he is investigating, and from these sections it is clear that the chief aggregation of lymphoid tissue in the submucous tissue of the stomach is in the pyloric region. A less extensive massing of lymphoid follicles is found in the cardiac fundus. Lymphoid follicles are only found at long and irregular intervals throughout the body of the stomach. In the pyloric regions these little rounded masses of adenoid tissue lie between the bases of the pyloric glands at regular and frequent intervals, and become more numerous as the pyloric sphincter is approached. In only one or two instances did Dr. Miller show me lymphoid follicles on the surface like a Peyer's patch, and then there was doubt that the mucous membrane had been removed by ulceration or post-mortem digestion. These lymphoid follicles are probably foci from which sepsis and acute ulcers of the stomach commence.

The lymphatic vessels are divided into three main groups, and roughly may be said to follow the veins in position and direction. Their surgical importance is that they determine the direction and limits of cancerous extension.

The most important group runs from the pylorus along the lesser curve, accompanying the coronary vein. These lymphatic vessels and glands are intimately connected with the stomach wall, and it is necessary to remove this as high as the point at which the coronary vessels reach it in order to effectually clear out cancer of the pylorus. These coronary lymphatics terminate in the cœliac glands in front of the aorta.

The second set runs from left to right along the greater curve, accompanying the right gastro-epiploic vein. This group is not in contact with the stomach wall, and to include it with the part removed in a partial gastrectomy it is necessary to remove rather more than an inch of the omentum attached to the stomach. These lymphatic vessels terminate in glands below the pylorus, and behind it around the head of the pancreas, and these must be carefully removed in cases of cancer.

The third set of gastric lymphatic vessels converge from the cardiac fundus and the left extremity of the greater curve, and pass through the gastro-splenic omentum to join the splenic lymphatic glands.

Another point of great surgical importance is that the lymphatics do not pass from the pylorus towards the duodenum, so that the section may be made only an inch beyond the obvious limit of a pyloric cancer ; whilst, from what has been already said, it will be clear that the section through the body must be made in a line from the œsophagus to a point to the left of the middle of the greater curve.

The nerves of the stomach are derived from the two vagi, and from the sixth, seventh, eighth, and ninth dorsal segments of the spinal cord through the sympathetic system.

As has already been pointed out, under the head of Development, the referred pains from the anterior surface tend to be on the left side, and those from the posterior surface on the right. Moreover, the cardiac orifice is innervated from the sixth and seventh dorsal segments, and the referred pain and hyperæsthesia from an ulcer in this region will be in the skin distribution of these segments. The pylorus is innervated by the ninth dorsal segment, and an ulcer in this region, as is commonly the case, will refer its pain to the corresponding skin segment.

The gastric glands are of two types, the pyloric and the cardiac. In the pyloric glands the duct leading to the secreting acini is much deeper. The secreting acini are several for each duct, and the cells lining them are all of one type, and are believed to secrete pepsin. In the cardiac glands the duct is much shorter. The secreting portion of the gland is usually formed of a single tubule, and is lined with central cells, similar to those in the pyloric glands. There are, however, in addition border or oxyntic cells which secrete the hydrochloric acid. These cells are most numerous in the glands of the cardiac fundus. The microscopic character of the gastric glands seems to have very little influence over gastric diseases. It is, however, stated that in advanced gastric cancer there is a general degeneration of all the gastric glands, and it is certain that hydrochloric acid and pepsin disappear from the gastric contents.

3. PHYSIOLOGY.

The physiology of the stomach is best considered under the heads of—

(A) Gastric movements.
(B) The secretion of gastric juice.
(C) Absorption from the stomach.

(A) **Gastric Movements.**—The movements of the stomach have been very fully investigated by W. B. Cannon (*American Journal of Physiology*, 1898, vol. i., p. 359), where a very full review of the literature of this matter will be found. Cannon's method was to feed cats on 15 grammes of bread and milk to which 5 grammes of bismuth subnitrate were added. He then watched the movements of the stomach by means of X rays and a fluorescent screen. Female cats were much more tolerant of the restraint of the cat-holder than tom-cats.

1. **Movements of Digestion** may be divided into (*a*) churning movements, and (*b*) the evacuation of the stomach contents.

(*a*) *Churning Movements.*—Cannon found that five minutes after a meal had entered the stomach peristaltic waves appeared as constrictions in the gastric shadow near the pylorus, and progressed slowly to the right. Gradually these constrictions commenced farther and farther to the left, until they all started from the transverse band—that is to say, a constriction about the middle of the stomach. This band, which is an anatomical feature of the stomach, becomes more and more tonic as digestion proceeds, but does not actually divide the lumen of the stomach in two until the end of digestion. To the left of the transverse band the cardiac part of the stomach acts only as a reservoir, and undergoes no change except a general tonic contraction. The pyloric part of the stomach to the right of the transverse band is more or less clearly divided into the pyloric antrum and the pre-antral part. When digestion is thoroughly established, a constriction starts every ten seconds from the transverse band, and in thirty-five seconds reaches the pylorus. There are always, therefore, three or four constrictions in transit in the pyloric part of the stomach. Cannon estimated that 2,600 peristaltic waves were employed by a cat in the digestion of an ordinary meal of bread and milk. These churning movements of the stomach are at once inhibited by pain or emotional excitement of any sort. As digestion proceeds, the pyloric part of the stomach becomes more and more elongated. The control of the pyloric sphincter is a most interesting matter; it is controlled partly from the stomach above, and partly from the duodenum below. Cannon gave his cats little buttons of hard-baked dough nearly solid with bismuth. He was able to watch these approach the pyloric sphincter and strike against it. The sphincter was then closed during several contractions, and no further chyme escaped into the duodenum. It has long been known that solid masses are not allowed to pass the pylorus until the end of digestion, when the stomach completely empties itself. This reflex is, no doubt, much more sensitive when an open ulcer exposes bare nerve terminals, and in these cases spasmodic contraction of the pylorus is almost constant.

It was Pawlow who found that an even greater control was exerted over the pylorus by the reaction of the duodenum, and this has been named "the acid reflex of Pawlow." Whilst digestion was actively proceeding he was able, through a duodenal fistula, to introduce acids and alkalies into the duodenum. He found that when acids were introduced the pylorus was closed, and no more acid chyme was admitted to the duodenum; whilst when the duodenal contents were rendered alkaline, nearly every gastric contraction forced chyme into the duodenum. This is, no doubt, due to the fact that in an acid medium pepsin will digest trypsin, and that, were the duodenum flooded with acid chyme, the whole of the pancreatic digestion would be disorganized. By the acid reflex the chyme is only admitted at just the rate at which it can be neutralized. There is also some evidence that mechanical distension of the duodenum closes the pylorus reflexly.

(*b*) *The Evacuation of the Stomach Contents.*—Pawlow found that, when the stomach was empty, at intervals peristaltic waves passed along it towards the pylorus. When food was admitted to the stomach, these movements of evacuation were inhibited, the pylorus contracted, and churning movements appeared. He further found that the administration of acids increased the pyloric contraction and retained the gastric contents, whilst alkalies, especially sodium bicarbonate, relaxed the pylorus, and led to rapid evacuation of the food in the stomach. It is clear from this that if we wish to increase gastric work we should give acids, whilst if our object be to save as much as possible an inflamed organ, we should soon after a meal administer an alkali, and thus shift the burden of digestion on to the pancreas.

2. **Movements involved in Vomiting.**—Cannon found that during vomiting the pylorus, transverse band, and the pyloric part of the stomach, were tonically contracted. No doubt this contrac-

tion is relaxed between the spasm of continued emesis, for otherwise it is hard to explain why gastric contents are so constantly followed in all persistent vomiting by bile-stained fluid. When the pylorus has been firmly closed, the cardiac sphincter is relaxed, and the contraction of the longitudinal fibres of the stomach draws the cardiac orifice open and disposes the cardiac reservoir in a convenient position below it for the expression of its contents. The actual ejectment of these contents appears to be effected by the simultaneous descent of the diaphragm above and the contraction of the abdominal wall below. These two muscular bodies squeeze the stomach between them, and force the contents up the œsophagus.

(B) **The Secretion of the Gastric Juice.**—Our present complete knowledge of gastric secretion we owe almost entirely to Pawlow. He has utilized in physiology the great advances in antiseptic surgery and the technique of œsophageal and gastric surgery to establish in dogs permanent œsophageal, gastric, and duodenal fistulæ, and to isolate from the main cavity a miniature stomach which retained its blood and nerve supply intact, and was connected to the surface by a separate fistula. Direct observation showed that the secretion of this miniature stomach accurately followed that of the larger cavity, and differed only in being about one-tenth in quantity. The great advantage was, of course, that the juice was not mixed with the food introduced into the larger cavity.

The discoveries of Pawlow and his pupils may be summarized thus: The secretion of gastric juice commences five minutes after the animal sees and desires food; and although the mastication and tasting of the food assists this flow, it is not necessary that the material should enter the mouth at all. This "psychic juice," as Pawlow named it, acts upon the food when it reaches the stomach, and elaborates from each article of diet a specific excitant, which acts upon nerve terminals in the mucous membrane, and leads to a second secretion of gastric juice, which is exactly adapted, in quantity and strength of ferment, to digest that particular kind of food. This second secretion is therefore the result of a chemical stimulation of secretion.

1. **The Psychic Juice.**—In order to demonstrate this secretion, Pawlow employed œsophagotomized and gastrotomized dogs. The food devoured and swallowed fell out of the fistula in the neck. The resulting secretion of gastric juice was collected from a tube placed in the gastric fistula, and was measured and

investigated as to its digestive power. It was found that the flow commenced about five minutes after food was offered to the dog, and varied in quantity, rapidity of flow, and digestive strength, with the desire the dog had for the food in question. Most curious and interesting differences were thus discovered. Not only was much juice secreted when the dog was hungry, and little when he was satisfied, but the flow varied according to the dog's palate. Thus meat excited a profuse flow, but bread and milk a much less secretion ; and even individual peculiarities, such as a special liking for certain kinds of food, invariably produced a corresponding change in the gastric juice dropping from the gastric fistula. The co-ordination even went so far that, when a dog had several times been teased in vain by the preparation of a tasty meal under his very nose, which was after all not given him to eat, he would at last learn the trick and turn away sulkily, and no juice or very little would then be secreted. This commencement of all gastric digestion by a purely psychic flow has, of course, a most important bearing on human medicine and health. For digestion to be complete and efficient, the man must hunger for his food, it must be of such a kind as he likes, and he must eat it with attention and relish. The notion that the value of a diet depends on the number of calories of energy its constituents contain is obviously absurd. For food, however nutritious in theory, which is abhorred, introduced without appetite, or even with repugnance, into the stomach, will lie there an inert mass for an hour or more and ferment. The importance of attention, appetite, and leisure, during meals is also obvious, and the bad habit of reading at meals must be strongly condemned.

2. **The Chemical Stimulation of Secretion.**—Pawlow investigated the manner in which the specific secretion for each article of food was brought about by employing dogs in whom a miniature stomach had been isolated from the main cavity. Whilst the dog slept, various articles of diet were introduced through a fistula into the main stomach unnoticed by the animal, so that no psychic secretion was excited. The secretion resulting was collected from the miniature stomach uncontaminated by the food.

Pawlow found that articles of diet so tested might be divided into four classes : (*a*) Those that produced a powerful secretion, (*b*) those that produced a moderate secretion, (*c*) those that produced no secretion at all, and (*d*) those that actually inhibited secretion.

The latter were discovered by mixing them with substances from classes (*a*) and (*b*), when they diminished or actually stopped the flow which would otherwise have resulted.

(*a*) *Substances which produce a Powerful Gastric Secretion.*—These are meat extractives, such as soups, broths, beef-tea, but especially Liebig's extract. The secretion commences in about a quarter of an hour. This interesting demonstration of the powerful action of these substances clearly explains why soup is taken before the modern civilized dinner, which is for the most part eaten without much appetite, desire, or actual hunger, and is usually far beyond the actual needs of the organism.

It also elucidates the great benefit derived from mixing beef-tea or meat extracts with the food when a patient is very ill and has not the least appetite for food, but the stomach itself is not diseased. When, however, the stomach is irritated, inflamed, or even ulcerated, meat extractives are obviously contra-indicated, for our object should then be to give the organ all the rest we can.

(*b*) *Substances which produce a Moderate Gastric Secretion.*—These are water, milk, and gelatine. It is not very clear why water should invariably produce a moderate and quite active secretion of gastric juice in the dog, unless it be that this fluid is so often dirty and contaminated that Nature has provided this gastric flow in order to kill and digest water-borne infections. Pawlow seems to have paid little attention to the antiseptic action of the stomach and duodenum; for it has long been known that, although a phthisical patient must swallow myriads of tubercle bacilli, these cannot be found in his fæces until his ileum becomes ulcerated, and this does not occur until dyspepsia is well established. The automatic and moderate secretion of gastric juice for milk is, again, of great interest; for the new-born babe cannot take much interest in its diet, and universal empirical experience has shown that there is no diet so nutritious as milk for those who are too ill to desire food or take it with appetite or attention. The empirical use of jellies as an important part of all invalid diets is also hereby explained by the automatic and moderate secretion gelatine excites.

(*c*) *Substances which produce no Gastric Secretion.*—These are egg-white, sodium chloride, starch, and sugar. These substances were, of course, given dissolved in water, and when it is said that they produced a negative result, it is meant that the secretion was no greater than a corresponding quantity of plain water.

That raw white of egg should, of all proteid substances, require no gastric juice for its digestion is certainly a most interesting fact, especially when it is remembered that for many years it has been used as an even more suitable and less exacting diet than milk in all cases of gastritis. The absence of all secretion in the case of starch and sugar is easily explained by the fact that gastric juice is much less adapted to the digestion of these than the pancreatic secretion.

(*d*) *Substances which inhibit Gastric Secretion.*—These are sodium bicarbonate and fats. We have already seen, under Movements of the Stomach, that sodium bicarbonate leads to a rapid evacuation of gastric contents through the pylorus into the duodenum, and to this we can now add that its administration checks further secretion, a most useful thing if it be excessive, and neutralizes such juice as has already been secreted, thereby rendering the pepsin inert. We have, then, in sodium bicarbonate a most useful means of terminating gastric activity whenever we wish it.

When Pawlow poured oil into the stomachs of his dogs, and soon after fed them on meat, no gastric juice appeared for half an hour, and then the flow was feeble and of weak digestive power. Considering that the secretion which failed to appear was the psychic juice, this inhibitory action must have been a reflex nervous one, acting on the glands or their secreting centre from the mucous membrane of the stomach.

We see many examples of this powerful inhibition in dietary. The familiar combination of bread and butter throws very little pressure on gastric digestion, which is restrained by the butter; but when the combination passes into the duodenum, the fat powerfully stimulates pancreatic secretion, and the bread is digested with it. Bread alone requires a small quantity of powerful juice, and butyric acid powerfully stimulates gastric secretion, probably because butyric acid fermentation is inhibited by hydrochloric acid. Bread and rancid butter, therefore, are the very antipathy of bread and fresh butter in their effect on gastric digestion. The worst effects of the inhibitory action of fat on gastric secretion are seen when it is combined with meat and administered to an enfeebled stomach. Perhaps the most familiar example of this is when fat mutton is forced on anæmic girls by austere parents, and produces severe dyspepsia and vomiting.

It is in such cases of weak gastric digestion that meat nearly fat-free, such as fowl and fish, should be employed. In cases of hyperacidity fatty emulsions may be used to restrain the secretion. Pawlow found that the moderate quantity and strength of the juice required to digest milk was due to the fat in it. When cream was added to the milk, the resulting juice was even further diminished and weakened. When fat was removed from the milk, the juice became more abundant and stronger.

To sum up the foregoing paragraphs: When an animal hungers and desires food, and more especially when his desires culminate in eating and swallowing food that he likes, the stomach by a reflex psychic act pours out gastric juice on that food within five minutes. This juice starts the process of digestion, and elaborates from each article of food specific substances which act upon the nerve terminals in the gastric mucous membrane, and direct the course of gastric secretion so that the juice is exactly adapted to the food to be digested.

It thus appears that each kind of food has its own particular kind of juice, and that these vary in quantity and digestive strength. In the same dog the same food always produces the same juice in the same quantity. If the food is doubled, the juice is doubled. From this it appears that in health secretion is most exactly adapted in quantity and quality to the work to be done, and there is a corollary to this, that in health there is no excess of juice secreted to digest and irritate the gastric wall. Gastric juice is far too corrosive a fluid for Nature to slop it about an alimentary canal in careless excess.

We may here enumerate a few of the varieties of juices which were secreted from the miniature stomachs of Pawlow's dogs when they were fed on special articles of food. In these the psychic and chemical secretion are combined, as the animal was allowed to devour the food which passed immediately into the main cavity of its stomach.

(*a*) Meat produced a large quantity of gastric juice of weak digestive power; (*b*) bread produced a small quantity of powerful secretion; (*c*) milk, a small quantity of small digestive power.

Milk is so important a food in disease, as well as in health, that its digestive characters may be reviewed and emphasized here.

In the first place, its digestion requires no psychic juice to start it. Taken into the stomach, it acts immediately upon the secretory nerves and produces its appropriate juice. It is therefore one of the only available foods for those who have no appetite, or who, prostrated by disease, take no interest in food or any other thing. Nearly all other foods would under these circumstances fall into the stomach, and lie there inert until they decomposed.

In the second place, the amount and strength of juice required to digest milk is very small, so that the least possible call is made on a stomach the powers of which are already greatly lowered.

In the third place, the proteid of milk is in a most available and easily digested form. If it be agreed that the increased secretion of urea after a meal is a gauge of the amount of proteid used up in the assimilation of the remainder, then in the case of milk 90 per cent. is assimilated, whilst only 50 per cent. of the proteid of breadth is available under the same circumstances.

One or two further facts alone remain to be stated. Pawlow found that gastric juice was always secreted at a constant acidity of 0·5 per cent. of hydrochloric acid. It is well known from test-meals that this acidity is reduced to a variable extent under different circumstances. No doubt this reduction is, in part at least, effected by the neutralizing action of the food; but probably there is some physiological means of reducing the acidity of the juice, when once secreted, to an optimum for the digestive processes proceeding. It is definitely known that an accumulation of hydrochloric acid stops the secretion of gastric juice.

When the stomach is empty, its mucous membrane, like every other mucous membrane, secretes an alkaline viscid fluid. Pawlow found that during attacks of gastritis in his dogs, even when empty, the inflamed stomach secreted a thin acid fluid. When we consider the etiology of gastric ulcer, we see that this observation is of great importance, for these alkaline intervals are probably of great importance for the repair of lesions of the gastric wall.

(C) **Gastric Absorption.**—Apparently the stomach absorbs very little. When its digestive work is done, or when it has none to do, it passes its contents rapidly into the duodenum, from the villi of which rapid absorption occurs.

The only direct experiment on this point of which I am aware is that of Dr. Edkins. He ligatured the pylorus in a cat, and then introduced into its stomach a many times lethal dose of strychnine, a drug to which cats succumb in the smallest doses. No ill result happened during several hours whilst the pylorus was ligatured, but as soon as the ligature was removed the cat passed into strychnine convulsions.

THE SIMULATION OF ACUTE PERITONITIS BY PLEURO-PNEUMONIC DISEASES*

THIS paper deals with a class of cases in which it is most easy to mistake thoracic for abdominal disease. This deceptive likeness of two diseases is not of purely theoretical interest, for a mistaken diagnosis is often followed in such a case by an unnecessary operation. This condition, whilst well known to the older physicians, has dropped out of notice in recent years, and that is some excuse for this paper.

CASE I.—**Right Diaphragmatic Pleurisy and Pneumonia with Marked Abdominal Signs, simulating Appendicitis with Peritonitis.**—This case I owe to the kindness of Dr. P. Kidd. A little girl aged eleven years was admitted to the medical wards of the London Hospital in 1895, complaining of pain in the head, but especially in the abdomen. Three days before admission she had severe pain down the right side of the abdomen, and vomited bilious fluid several times. When she was first seen at the hospital, her abdomen was rigid and especially tender in the right iliac fossa. Her bowels had not been opened for three days, and her tongue was brown and dry. Her pulse was 104, full, and not easily compressed, and her temperature was 103° F. The thoracic signs, which at no time were very marked, were almost absent on admission; she was, however, breathing about forty to the minute, and cut her respirations short because they hurt her in a manner characteristic of pleurisy. She had, moreover, tenderness along the attachment of the diaphragm, and the veins were dilated there. By the fourth day after admission there were obscure signs, at the right base, of some pneumonia, the breath-sounds were a little diminished, and there were a few moist râles; but these were also heard at the left base. Her temperature, which had remained at about 103°, fell by crisis to normal on the fourth day, but soon became irregular, and for four or five weeks it frequently reached 102°. During this time the signs of pneumonia and diaphragmatic pleurisy became more distinct, and at last left no doubt as to her condition; but had she been admitted to a surgical instead of a medical ward, and at the present time, when so many cases of appendicitis are submitted to early operation, instead of seven years ago, I cannot help thinking that her abdomen would probably have been explored.

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* Reprinted from *The Lancet*, August 2, 1902.

CASE. II.—**Pleuro-pneumonia of the Right Base; Acute Abdominal Pain and Vomiting; No Lung Signs; Herpes Labialis; Empyema. Recovery.**—This case I owe to the kindness of Mr. H. P. Dean and Dr. G. Schorstein. A little boy aged ten years was admitted to the London Hospital in 1899 as suffering from acute peritonitis after an injury. Two days before admission he had received a blow in the lower part of the chest or in the upper part of the abdomen. On the following day, the day before admission, he was seized with severe pain in the abdomen, chiefly in the upper part, and he vomited twice. On admission, his abdomen was found to be somewhat distended, tender, and rigid all over, but especially so in the epigastric region. His bowels had been constipated since the pain commenced, and an enema only gave a small result. He lay with both legs drawn up. His temperature was 99·5° F. on admission, but it rose to 103° the same evening. His pulse ranged from 130 to 140, but was of good volume and strength. His respirations were forty-four per minute and catchy. The examination of the thorax gave absolutely negative results. Soon after admission he developed herpes labialis, and was transferred next day to the medical side. No signs had yet appeared in the thorax, but on the following day there was undoubted consolidation at the right base. The temperature fell by crisis on the ninth day of the disease, but seven days later it rose again and became irregular, and empyema developed on the right side. This was opened and drained, and he then made a good recovery.

CASE III.—**Double Basal Pneumonia with Right Diaphragmatic Pleurisy in a Girl the Subject of a Gastric Ulcer; Acute Epigastric Pain with Collapse. Laparotomy; Death.**—This case is certainly the most perplexing one that I have yet heard of; I am indebted for it to Mr. F. S. Eve. A girl aged seventeen years had been attending the out-patient department of the London Hospital for some months, for marked anæmia and gastric ulcer. She had had once before a similar attack to the one which I am about to describe. One morning in 1899 she started for work as usual, and on her way was seized in the street with violent epigastric pain and vomiting. She was brought to the hospital at once in a state of collapse. Her pulse was 120 and very thready, and her temperature was 104·5° F. Her abdomen was rigid, motionless, very tender, and distended, and these signs were most marked in the epigastric region. She was admitted to the surgical wards as a case of ruptured gastric ulcer, and within two hours Mr. Eve opened her abdomen. He explored the anterior and posterior surfaces of the stomach, the greater and lesser curvatures, but found no trace of gastric ulcer, nor was there any peritonitis. Unfortunately, ether was the anæsthetic given. Misfortunes followed fast. Her cough became so violent that a coil of intestine and some omentum escaped between the stitches, and had to be washed and returned and the abdomen again sewn up. On the second day after admission it was clear that she had right basal pneumonia, but her temperature had fallen to 101°. On the third day it rose again to 104°, and signs of consolidation appeared at the left base, and she died on the fifth day from the commencement of the attack. At the post-mortem examination double basal pneumonia and right diaphragmatic pleurisy were found, and in the stomach was a shallow ulcer of the size of a sixpenny-piece, which was not even near perforation. There was no peritonitis. I think that all will admit that this was an impossible case to diagnose correctly. Looking back at the case, the only point which should have aroused suspicion was the temperature of 104·5°. No record of the frequency of respiration was kept.

CASE IV. — **A Case of Pyopneumothorax of the Left Base simulating Appendicular Peritonitis or Left Subphrenic Abscess of Gastric Origin.** — A male Hebrew aged thirty-four years was admitted to the London Hospital under a medical colleague for "colic and peritonitis" in 1901. I was asked to give a surgical opinion on his abdominal condition. He had not been well for a year, suffering chiefly from pain and wind in the stomach after food, but without vomiting. For several weeks he had also had pain in the left lower and back part of his chest, with a bad cough and night-sweats and wasting. He had been constipated for twelve days. When I saw him his temperature was 101° F., his pulse was about 100 and fairly strong, and his respirations were thirty-eight, his nares being vigorously dilated at each inspiration. He was a little icteric, but not really jaundiced. His abdomen was rigid and very tender all over, but I could make out no local dulness or resistance, and the abdominal wall relaxed between each respiration. The base of the left lung behind was dull on percussion. Vocal resonance and tactile vocal fremitus were diminished. Such breath-sounds as could be heard were of a somewhat tubular character. The house-physician thought that the right iliac fossa was more rigid and resistant than the rest of the abdomen. The medical registrar was of opinion that the signs indicated left subphrenic abscess due to a perforated gastric ulcer, but the stomach resonance was normal and the spleen was not displaced. My medical colleague agreed with me that the signs appeared to indicate trouble above the diaphragm, and arising from the lower lobe of the left lung. Ten days later it was clear that he had pyopneumothorax at the left base, probably arising from a tuberculous focus in the apex of his left lower lobe. A rib was accordingly resected, and his temperature fell to normal. He put on flesh and lost all pain, but a sinus persisted when he left the hospital.

CASE V. — **A Case of Pyopneumothorax of the Right Base simulating Appendicular Peritonitis.**—On September 12, 1901, I was called late one evening to see a man in the wards of the London Hospital, who was said by my house-surgeon to be suffering from "appendicitis and general peritonitis." Two days before admission the patient was running for a train on his way to work in the morning, when he was seized with severe pain in the right iliac fossa. He gave up work at 1 p.m. on the same day and took to his bed. Next day his bowels were well opened by a pill and castor-oil. He had no vomiting. He had never been seriously ill before. When I saw him in the wards, it was clear that he was seriously ill and in great pain. His temperature was 100° F., his pulse was eighty, his respirations were forty-three, and his tongue was furred and dry. His abdomen was hard, rigid, and tender all over, but perhaps a little more so in the right iliac fossa. It relaxed, however, between the respirations for a second, and became soft, and there was no marked distension or localized dulness or resistance. The heart was normal, and the examination of the lungs showed nothing but a little bronchitis and emphysema. I refused to operate because, whilst the abdominal signs appeared to me at least doubtful, the respirations—forty-three a minute, with a pulse of only eighty—appeared to indicate that the lungs were the source of the trouble. The thing, however, which really decided me not to operate was that I did not think that he could safely take an anæsthetic. I carefully examined him again on the next day with a medical colleague. The abdominal signs had disappeared almost entirely, and dulness at the right base, with diminished breath-sounds and tactile vocal fremitus, the presence of a pleural rub, and the absence of tubular breathing, left no doubt that he had right basal pleurisy. Eight days later the signs at the right base had much increased, and pus was found by an exploring needle. A rib was resected, and 24 ounces of pus with air were evacuated. The condition was then found to be one of pyopneumothorax. I saw him once again in February, 1902. He had been attending the out-patient department for a sinus left by the empyema. An energetic dresser had syringed out his cavity with some antiseptic, and had nearly choked him by the passage of the fluid through the orifice in the lung into the trachea.

CASE VI.—**Traumatic Left Pleurisy with Abdominal Signs. Laparotomy**.—A labourer aged thirty-eight years was admitted to the London Hospital under my care in February, 1901, giving the following history: He was assisting to unload deal planks from a barge, when one of the planks swung round suddenly whilst hanging from the crane, and struck him violently over the lower left ribs, precipitating him 12 feet into the barge, where he alighted on the back of his head. He was slightly concussed and had a scalp wound in the occipital region. He soon recovered from the shock of the fall, and the only sign of the injury which could be made out was a fracture of two of his left lower ribs, probably the ninth and tenth; but he was so muscular a man and the part was so tender that the exact ribs broken could not be determined. Within twelve hours of admission a pleuritic rub was heard in the region of the broken ribs. On the next day acute abdominal signs appeared. He complained of violent abdominal pain, which was worse down the left side of the abdomen. His abdomen was rigid all over, but especially in the left hypochondrium and lumbar regions; the abdominal wall, however, still moved on respiration. Ill-defined dulness was found down the left side, and this was thought to be due to blood in the peritoneum. He vomited continuously, and the vomitus soon became bilious. His bowels had not been opened since he was admitted, and an enema of 1½ pints of soap and water was given. This was retained. Retention of urine, which had been present since admission, persisted. His temperature was 99° F. and his pulse was 75, and of good volume. A diagnosis of laceration of the spleen with recurrent hæmorrhage was made, and laparotomy was decided upon. I must admit that this diagnosis was largely due to the fact that within a fortnight a very similar case had been admitted to the same ward, and did not develop any marked abdominal signs until the fifth day, when the patient was to have left the hospital. On that day he collapsed, and his abdomen became distended and much free fluid was discovered. Laparotomy was performed, and the spleen was found to be torn nearly in two. He died from hæmorrhage on the table whilst the clots were being displaced to find the pedicle. The abdomen in the present case was opened in the left linea semilunaris and the abdominal contents were found to be absolutely normal. The dulness was explained by the enormous muscular development of the abdominal wall, which was so great that the recti and lateral abdominal masses projected into the peritoneum as rounded masses, which were at first thought to be hæmatomata. The great muscular development of the abdominal wall was apparently due to the man being by trade a deal-runner. Some idea of the spasm may be gained by the fact that strand after strand of ordinary silkworm gut snapped without drawing the edges of the incision together, and finally the stoutest silk had to be employed and the anæsthetic vigorously pushed before the abdominal wound was closed. The wound healed by first intention and the patient made an uninterrupted recovery.

Andral* appears to have recognized this condition clearly in 1836, and in his book narrates several typical cases. Watson,† in his admirable textbook of medicine, quotes Andral, and refers to cases of his own in which pleurisy at the right base simulated hepatitis; but he does not appear to have recognized the possibility of confusing diaphragmatic pleurisy with acute peritonitis. Fagge knew the condition well. In his textbook he says: "In exceptional cases the pain is referred to the terminal branches of the intercostal nerves; to the hypochondrium, so as to lead to a mistaken diagnosis of hepatitis; to the loins, so that the case has been called one of lumbago; or to the neighbourhood of the umbilicus, so that peritonitis has been suspected;" and in a note he adds: "I have myself had a patient whose sole complaint was of pain in the crista ilii. I feel sure that, if it had not happened that a short time before my attention had been specially directed to this question, I should have failed to discover that he had pleurisy, although on applying my stethoscope I at once heard a rub.—C. H. F."* Modern medical writers for the most part are vague in their reference to this class of case, and many do not even refer to them at all. The condition is altogether unknown to surgeons if we judge from their textbooks, and this is all the more unfortunate, as it is they who will open the abdomen of the pleuritic patient should they be ignorant of this condition, after they have caused ether to be administered. My excuse for this paper is to call attention to a pitfall in surgery into which, as may be seen here, I have fallen myself.†

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* Andral, translated by D. Spillane, 1836.

† Sir Thomas Watson, "Principles and Practice of Physic," fifth edition, 1871.

The thoracic diseases which can give rise to such severe abdominal signs as to simulate peritonitis are all situated in the lower part of the chest, either in the base of the lung or the adjacent pleura—diaphragmatic or parietal. Three of my cases were those of pleuro-pneumonia of the base of the lung, involving the diaphragm. Two were cases in which apparently a tuberculous focus in the apex of the lower lobe ruptured into the pleural cavity, producing pyopneumothorax. One case was most interesting in that the pleurisy was traumatic, due to a localized injury, and associated with broken ribs; it was recognized, and yet the abdomen was opened, so definite were the abdominal signs.

Signs and Symptoms.—The abdominal signs and symptoms which may arise in such conditions are conveniently divided into parietal and visceral.

Parietal.—The abdominal wall is acutely tender and painful. The tenderness often extends over the whole abdomen, but in some cases it is unilateral; it may be present only above or below, or be more intense in these situations. It will readily be seen that such acute abdominal pain and hyperæsthesia in the right hypochondrium may be mistaken for hepatic and biliary diseases, whilst in the right iliac region an appendicitis would at once suggest itself, and in the epigastrium a perforated gastric ulcer. The abdominal muscles are, moreover, in spasm, so that the abdominal wall is board-like and hard. So great is this spasm that the deepest anæsthesia is necessary to relax it, and the pupils will be widely dilated before the abdomen becomes soft. This has practical application, for if the intestines are allowed to escape, they will be returned to the abdomen with the greatest difficulty, and the surgeon will find that the stoutest silk alone will stand the strain necessary to close the wound. The respiration is of the costal type, but it will be found, on placing the hand upon the abdominal wall, that this relaxes for a moment at each inspiration, and this is an important diagnostic sign. Writers on diaphragmatic pleurisy insist on the importance of unilateral exaggeration of costal respiration on the side of the chest affected.

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* Hilton Fagge, "Principles and Practice of Medicine," third edition, i. 1026.

† Since this paper was read at the Clinical Society on April 11, 1902, a paper has appeared on "The Simulation of Appendicitis by Acute Intrathoracic Disease," by Dr. Maurice H. Richardson, *Boston Medical and Surgical Journal*, April 17, 1902.

Visceral.—The visceral signs are distributed along the whole length of the alimentary canal. Gastric disturbance leads to nausea, hiccough, and vomiting, which may be urgent and become bilious. The intestines are apparently more or less paralyzed, so that the abdomen becomes distended, and this, with the tenderness and spasm of the abdominal wall already referred to, produces a most deceptive resemblance to peritonitis. Constipation is nearly always present, and enemas may be retained, so that apparently the rectum has lost its irritability. Even the profound vascular changes which we are in the habit of associating with severe lesion in the splanchnic area are frequently present, at least at the onset of the attack, although it must be admitted that they do not often persist or become more severe as the disease develops.

The patient is often collapsed at the onset of the pain. The extremities are cold, and beads of sweat may break out on the forehead, whilst the features are pale and pinched; but in none of my cases did I see the rings round the eyes, or the dry, shrivelled tongue, which one associates with general suppurative peritonitis.

Diagnosis.—The diagnosis is nearly always easy if suspicion is aroused and the surgeon is aware of this deceptive condition. The chief points to be observed are the rapidity of the respiration, often forty a minute or more, which is out of all proportion to the pulse-rate, which is seldom so frequent as 100 a minute, and is not weak or wiry. The respirations will often exhibit the curious catch at the top of inspiration which is characteristic of pleurisy.

The abdominal tenderness is, moreover, found to be chiefly superficial, and firm deep pressure with the flat hand may be permitted. It will then be found that the abdominal wall becomes soft for a moment at each inspiration, which is not the case in acute peritonitis. The absence of rings round the eyes, and a temperature of about 103°, should also make one hesitate to diagnose a serious and general peritoneal infection. When once a suspicion has dawned on the mind of the surgeon, the patient is comparatively safe, for an examination of the suspected side of the thorax will reveal some dulness or friction sound, or some excess of costal movement, on that side over the other, which will lead him to pause, and perhaps to consult his medical colleague; but it must be frankly admitted that direct thoracic signs are often almost entirely lacking for twenty-four hours or more.

The great danger, after all, appears to arise from an autosuggestion by the patient's surroundings and the circumstances under which he is first seen. The surgeon is summoned by his house-surgeon, perhaps in the dead of night, to see a case of "acute peritonitis probably of appendix or gastric ulcer origin." He goes to his well-known ward, where he has seen series of cases of every phase of abdominal disease, but seldom or never pleurisy or pneumonia. There he finds a patient suffering from acute abdominal pain, perhaps vomiting, and the house-surgeon tells him that the patient has retained an enema. The pain the patient is in, and perhaps his cries when he is being examined, disturbing the whole ward as they do at night, prevent a complete examination. It may be that the surgeon is not quite satisfied that the signs undoubtedly indicate general peritonitis, but his clinical experience at once reminds him how vague the signs of general peritonitis may be; of the case of ruptured liver or spleen in which the signs were so vague for three or four days that operation was postponed until too late; or of that case of ruptured gut which for two days was supposed to be abdominal contusions. In such circumstances it is really not surprising that patients exhibiting the signs which I have mentioned are sometimes operated on. This is an accident always to be deplored, for very frequently ether is administered, and this adds bronchitis to the pre-existing lung condition, whilst an extensive abdominal incision seriously hinders coughing. The only guard against such deplorable accidents is to recognize the possibility of their occurrence, and that is my excuse for this paper and the record of these cases.

Pathology.—The pathology is one of referred pain, and divides itself into three heads.

1. *Irritation in continuity* of the lower six dorsal nerves will explain the hyperæsthesia and spasm of the abdominal wall. These intercostal nerves supply the diaphragm and lower part of the pleura, and here their branches, if not their trunks, will be irritated by an acute inflammatory process. The terminations of the seventh to the eleventh dorsal nerves pass to the abdominal wall, and supply the skin and muscles there. If the seventh, eighth, and ninth nerves are chiefly affected, the acute epigastric tenderness and spasm will closely simulate a perforated gastric ulcer; or if chiefly hypochondriac and on the right side, hepatitis or gall-bladder trouble. When the tenth dorsal nerve is mainly affected, the pain is referred to the umbilicus, as it is in so many abdominal complaints, whilst the area of the tenth and eleventh dorsal nerves on the right side is that in which the signs of an acute appendicitis develop. Moreover, the presence of tenderness and spasm on the left side in the tenth and eleventh dorsal areas does not exclude appendicitis, for in the early stage of this affection the pain may be referred to that side. It is interesting to note that three of my six cases terminated in suppuration. This seems to indicate that the inflammation, in order to produce such abdominal signs, must be severe enough to penetrate to the nerve trunks themselves and to produce actual neuritis.

2. *Referred visceral pain* has been most exhaustively studied by my colleague Dr. Henry Head,* and it will explain referred abdominal pain from lung disease without pleurisy. The seventh, eighth, and ninth dorsal rami communicantes are those which supply the lungs. When broncho-pneumonia, phthisis, or bronchitis, but not lobar pneumonia, supervene, even without pleurisy, impulses may pass up to the posterior root ganglia of these nerves, and there produce such changes that normal impulses passing through these ganglia from the terminal branches of the nerves in the epigastrium and upper abdominal wall are exalted into painful impressions. The spasm follows in a reflex manner from the pain. Dr. Head tells me that he has seen such cases, but none of those related here are explicable on this pathology.

3. *Of the pathology of the visceral signs* to which I have referred, gastric, intestinal, rectal, and vascular, we know very little.

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* "Herpes Zoster," *Brain*, 1900; "Referred Visceral Pain of Heart and Lungs," *Brain*, 1896.

Perhaps they are the result of a reflex in the reverse direction to that to which I have just alluded, so that irritation of the six lower intercostal nerves by a pleurisy produces disturbance in the distribution of the three splanchnic nerves which are derived from the same level of the spinal cord. On the other hand, it may be due to irritation of these nerves in continuity as they lie beneath the pleura or pass through the diaphragm to the great sympathetic plexures and ganglia of the abdomen.

THREE LECTURES ON ACUTE APPENDICITIS

LECTURE I

THE ETIOLOGY, PATHOLOGY, AND SYMPTOMS, OF APPENDICITIS IN RELATION TO ITS TREATMENT*

GENTLEMEN,—In this lecture, which is the first of three, we shall consider the causes of appendicitis and such of its symptoms as will guide us in the vexed question between medical and surgical treatment. Next week we shall deal with appendix abscesses.

It has always appeared to me a deplorable thing that, when a case of appendicitis has passed through the dangers attending its acute stage in the first week, it should after all succumb to the complications attending the formation of an abscess. In my third lecture I shall deal with general peritonitis, and I hope then to be able to tell you of a course of treatment which we are adopting at the London Hospital which appears to give promise of more hopeful results in this terribly fatal complication.

It is usual to consider an acute attack of appendicitis as the result of quite recent causes. On the other hand, I wish to impress upon you to-day that the conditions which have led to this acute attack have been in process of development for months, or perhaps for years, and that when the circumstances which make the first attack possible have once come into existence, the occurrence of subsequent attacks is to be expected. It has been truly said that in the affairs of men all great events are determined by the coincidence of the man and the hour; and in pathology it is equally true that acute inflammations are produced by the advent of the micro-organism and its opportunity. The micro-organism in the case of appendicitis is nearly always the *Bacillus coli communis.* The interesting point about this bacillus is that it is a normal inhabitant, not only of the human intestine, but of that of nearly all mammals. Yet it is only on very rare occasions that it produces pathological results. This fact is accounted for by the enormous differences which this bacillus exhibits in its virulence. This micro-organism obtained from the normal intestine is found to be of very low virulence when inoculated into animals. When taken from an intestine attacked by catarrhal inflammation it is found to be of much greater virulence; and, finally, when derived from cases of abscess and acute peritonitis it is of extreme virulence. This fact is of fundamental importance in the etiology of appendicitis, and we must consider and teach our patients to avoid the conditions which produced these changes in virulence of the colon bacillus. It is held by some pathologists that it is the catarrhal or inflammatory condition of the alimentary canal which increases the virulence of the *Bacillus coli.* On the other hand, some hold that the increased virulence of the micro-organism produces the inflammatory change; but however this may be, the fact remains that, when the alimentary canal is attacked by gastro-enteritis, the *Bacillus coli* in that alimentary canal is vastly increased in virulence, and is far more potent to attack the point of least resistance—the appendix. Other micro-organisms are occasionally found in connection with appendicitis. First we must notice the *Staphylococcus aureus*, and more rarely the *citreus* and *albus.* The *Streptococcus pyogenes* is sometimes found, and very commonly the *Bacillus aerogenes capsulatus.* This latter micro-organism was first described by Welch, of Johns Hopkins University. As its name indicates, it produces large quantities of gas, and is responsible for the occurrence of emphysema and of the distension of some abscess cavities with gas. This fact has a practical application, for in some cases you will find that although the other signs lead you to suppose that you have to deal with a large abscess, yet on percussing this part of the abdomen it is not only resonant, but even tympanitic, and when opened a very considerable quantity of gas will escape with the pus.

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* Delivered at the Polyclinic on February 20, 1903.

The typhoid bacillus has also been found as a cause of appendicitis, and a typhoid ulcer has even been located in the appendix. Still more rarely the *Amœba coli* has produced appendicitis in the course of dysentery, and in a very few cases appendicitis is due to infection with actinomycosis. Some years ago I encountered such a case, where the cæcum and appendix were welded together in a large indurated mass, and although it is impossible to state definitely whether the actinomycosis commenced in the cæcum or appendix, they were certainly both involved at the time of the operation. This case recovered after incision and drainage and the exhibition of huge doses of potassium iodide.

But if the micro-organism be the real cause of appendicitis, it is powerless to produce it without certain conditions being present which we have called its opportunity. Four of these conditions are of common occurrence; they are—(1) The presence of a foreign body in the appendix; (2) the existence of a stricture or kink; (3) an ulcer; or (4) the rich supply of lymphatic tissue in the walls of the appendix, which has led Mr. Bland-Sutton to style it the abdominal tonsil. The rarer conditions which give the micro-organism its chance of attacking the appendix are—(5) trauma, which may be direct or due to the violent action of muscles; (6) the terminal character of its blood-supply; and (7) in women the occurrence of menstruation. We will consider each of these causes in turn.

1. **Foreign Bodies.**—In 60 per cent. of fatal cases of appendicitis a foreign body is found in the appendix. In most cases this foreign body is a small, elongated, friable mass of what at first sight appears to be fæcal matter. When pinched between the finger and thumb it is easily crushed, and it is certain that, if its constituents be examined, small particles of undigested food will be found; but it is now generally held that a greater part of the material of which it is formed is produced by the inspissation of the secretions of the inflamed appendix.

More rarely we find a definite calculus in the appendix. These calculi are stony hard and covered with little, sharp, nipple-like projections. They are formed of triple phosphates with some organic matter. I have here several such calculi. One, you will see, is strangely like a date-stone; it was removed from a man who had suffered from many attacks of relapsing appendicitis. Others, you will see, are much larger and more irregular in shape. Their size makes it impossible that they can have entered the appendix from the cæcum. The wall of the appendix is greatly thickened and condensed around them, and the mucous membrane, you will see, is ulcerated in several places.

True foreign bodies are occasionally found in the appendix. We have here the cæcum, ileum, and appendix, of a man who died recently in the London Hospital of general suppurative peritonitis. The end of the appendix is enlarged into an irregular bulbous mass, and projecting from a small gangrenous spot you will see the point of an ordinary pin. I have also seen a grapestone removed from an appendix, and on another occasion a small shot, which no doubt had been swallowed while the patient was eating game.

Recently a good deal of attention has been called to the causation of appendicitis by worms locating themselves in the appendix. These worms are usually the *Oxyuris vermicularis*, whose normal *habitat* is the cæcum and colon. As a cause of appendicitis they must be extremely rare, but a friend of mine recently informed me that he saw an appendix removed for recurrent appendicitis which contained a mass of these little worms twisted together.

These foreign bodies act by harbouring in their substance micro-organisms, and by the mechanical irritation which they produce upon the adjacent mucous membrane. They also obstruct the lumen of the appendix, and lead to the retention of secretions in the part beyond.

2. **Strictures and Kinks.**—The lumen of the appendix is often obstructed at or about its base by strictures formed of inflammatory tissue in the submucous layer. In some instances the obstruction is produced by a sharp kink in the appendix, which appears to be due to adhesions in some cases, whilst in others we must rather regard it as a congenital deformity or as a result of the appendix being doubled up in the groove between the cæcum and the abdominal wall. In whatever way the obstruction is produced, the result is the same. The secretion of the appendix accumulates in the part of the lumen beyond the obstruction. and dilates it into a considerable cavity. At first the contents of this cavity are clear, gelatinous mucus, and such an appendix is known as a "mucocele." In the progress of time this mucus becomes infected with micro-organisms from the adjacent cæcum, and we may then find the dilated appendix filled with pus and its wall ulcerated. This is known as "empyema of the appendix."

3. **Ulcers.**—Ulcers are often found in the mucous membrane of an appendix; for the most part they occur in connection with the two previous causes—either a foreign body has irritated the wall of the appendix and produced an ulcer, or the retention of decomposing secretion has led to the same result. But in a few cases ulcers appear to arise independently of these conditions.

It is, of course, obvious that such an open sore in the appendix only requires the arrival of the virulent *Bacillus coli* to lead to acute inflammation of the entire structure.

4. **The Lymphatic Structure of the Appendix.**—It is an almost universal custom to speak of the appendix as a vestigial organ, and it is very generally supposed that it is the atrophied end of a cæcum which was very much longer in a previous stage of human evolution. This opinion appears to be incorrect. What its function may be is not known, but it is originally developed in connection with a mass of lymphoid tissue in the wall of the cæcum. This mass of lymphoid tissue is found in all mammalia, and it is as highly developed in the human being as in any other animal. Mr. Bland-Sutton was one of the first to draw attention to the lymphatic character of the appendix, and from the many close similarities which it presented he styled it the "abdominal tonsil." The submucous tissue of the appendix is always rich in lymphatic tissue, and its end is sometimes converted into a solid mass of lymphoid tissue not at all unlike a lymphatic gland. The appendix will be liable to attacks of acute inflammation in just the same way as the tonsils or the lymphatic glands in the neck, and that just as in children we see acute lymphatic abscesses arise in the neck after a slight catarrhal inflammation of the throat, so an acute suppurative appendicitis may arise from a catarrhal inflammation of the cæcum, or of the mucous membrane lining the appendix itself. I have here a specimen in which the appendix is swollen, congested, and violently inflamed, and yet you will see that it contains no calculus, and that there is no sign of a stricture or an ulcer in its lumen.

Turning now to the rarer causes of appendicitis:

5. **Trauma.**—When a disease, such as hernia, is much more common in men than in women, there is a strong supposition that it is due to the more laborious employment of the former. Now, appendicitis is four times more frequent in men than in women, and we may therefore suspect that it is often produced by some traumatism. In a few cases the injury to the appendix appears to be direct. There has been some blow upon the abdomen, or the patient has been run over, and the attack of appendicitis supervenes immediately. I have here a specimen which was obtained from such a case. A little boy of ten years old was thrown down in the playground and his abdomen trampled on.

He almost immediately complained of acute pain, and the symptoms of general peritonitis rapidly supervened. At the operation no coil of ruptured gut could be discovered, nor were any intestinal contents found free in the peritoneum. The peritoneal cavity was drained as well as his very serious condition would permit, but he died soon afterwards. This specimen, which was obtained at the post-mortem, shows a very large, hard, irregular calculus lying in an ulcerated cavity in the appendix, and you will see at one part a small perforation which produced the peritonitis. We can scarcely believe that this was an example of coincidence.

In another case of mine, a man aged thirty-five was kicked in the right iliac fossa by a horse. He vomited fifteen minutes later and developed peritonitis. Some days later a localized appendix abscess was opened, and he recovered. I must, however, confess that the appendix was not actually identified.

Appendicitis very frequently comes on suddenly when the patient is making some unusual or violent effort. A man is lifting some heavy object, when he is seized with a violent pain in the right iliac fossa. Several examples have been recorded from the football field. These instances have always been attributed to the action of the psoas muscle. It is supposed that the appendix has become adherent to the sheath of this muscle in some former slight attack, and that, when the muscle during the violent movement is contracted to an unusual extent, these adhesions are torn across and the lumen of the appendix is opened. No doubt this explanation is correct in many cases, but I wish to offer another possible explanation, and I will tell you how my attention was drawn to the matter. One day, whilst performing an appendicectomy, when the appendix had already been removed and its stump was secured in the field of operation by a pair of Spencer Wells' forceps, the anæsthetist allowed the patient to vomit. The root of the appendix was immediately drawn upwards and to the left into the abdomen, a distance of about two or three inches. I immediately pulled it back into the field of operation, and then, as the vomiting continued, I found that with each spasmodic effort of the patient a very considerable pull was exerted upon the root of the appendix. Now, this was not due to any contraction of the psoas muscle, for the thigh was not flexed by the effort. The true explanation is, I believe, as follows: The root of the mesentery, as you will see from this diagram, passes from the left crus of the diaphragm obliquely downwards and to the right across the spine, and terminates at the ileo-cæcal junction. We are aware that the mesentery has very considerable power of contraction, due to the presence in it of the muscle of Lockwood, which is prolonged as the muscle of Treitz into the left crus of the diaphragm ; and in violent efforts it is certain that the whole of the mesentery with the attached intestines is drawn up in the abdomen. Professor Arthur Keith attributes this elevation of abdominal contents rather to the broad band of the transversalis muscle which surrounds the abdomen like a belt. If, then, the appendix becomes attached to the lower extremity of the mesentery, and anchors it by adhesions in the right iliac fossa, these adhesions will be liable to be torn during the sudden elevations of the abdominal contents in violent efforts.

6. **Terminal Blood-Supply.**—It is a very common experience to find, in what appears to be a first attack of appendicitis, that the acute symptoms have been produced by sloughing of a greater or less piece of the end of the appendix, and that yet the calculus is situated about the middle of the organ, and that the tissues around this calculus have a good blood-supply and have not lost their vitality. From such cases we must conclude that the chronic inflammation set up around the foreign body has led to the obliteration of the vessels running to the part beyond, and that the final thrombosis of these vessels has determined the acute attack. In the work of Fowler, already referred to, there are several beautiful figures of vessels in the mesentery of the appendix in which this obliteration is going on. It is interesting to note, however, that the appendicular artery runs in the free margin of the mesentery of the appendix, and is therefore as far removed as possible from this source of danger. But the mesentery frequently reaches no more than halfway along the appendix, and the artery to the terminal half must then lie in actual contact with the tube exposed to the danger of thrombosis.

7. **Menstruation.**—This is a predisposing cause to which much attention has been directed lately. It is a matter of common clinical experience that attacks of relapsing appendicitis in women are frequently determined by the onset of menstruation. This introduces a difficulty into the diagnosis, for if the attacks be slight and accompanied by insignificant pyrexia, the symptoms may easily be due to the congested condition of the right ovary. This connection between menstruation and appendicitis has a most interesting anatomical basis. You will see in this specimen

stretching between the right ovary and the appendix, a well-marked fold of peritoneum which contains a branch of the ovarian artery to the appendix and many lymphatics. It can therefore scarcely fail to happen that, if the appendix be in a condition which predisposes it to inflammation, more or less acute symptoms should supervene when the ovary becomes greatly congested during menstruation.

You will at once perceive that all these conditions which we have spoken of as forming the opportunity for the micro-organism to attack the appendix were not produced in a day, but required for their development months, or even years, of preparation. We must, therefore, now consider those insidious and almost symptomless circumstances which lead to the production of foreign bodies, strictures, or ulcers.

First and foremost among these conditions is the narrow lumen of the appendix. In all parts of the body we find that narrow passages, more especially if they be blind, are liable to harbour micro-organisms and to become the seat of chronic inflammation. Thus the follicles of the tonsil become choked with secretion, and lead to chronic follicular tonsillitis with enlargement of lymphatic glands in the neck. The lacunæ of the urethra and the glands of the prostate harbour the gonococcus for years, and in a similar way the narrow, long, and tortuous lumen of the appendix is an ideal place where micro-organisms may lurk and secretions may accumulate. This produces a chronic catarrhal inflammation, which, as in the case of the urethra, may easily lead on to stricture when chronic inflammation has once been started in the submucous tissue; or the excessive secretion may become inspissated into a coprolith around some aberrant food particle.

It is almost certain that a chronic catarrhal inflammation of the cæcum precedes that of the appendix. This inflammation in many recurrent attacks invades the narrow lumen of the appendix, and in each of these, as the mucous membrane swells, the lumen is obstructed in the same manner as catarrhal inflammation of the duodenum, spreading up the common bile-duct, occludes its lumen and produces catarrhal jaundice.

Now, what are the causes which may lead to this chronic inflammation of the cæcum? They are believed to be due for the most part to irregular habits, to meals bolted in haste and without any leisure for digestion afterwards, to an irregular emptying of the bowel; but it is curious that appendicitis does not appear to be due to constipation, for if it were we should expect it to be much more common in women and in older people than in the young. The disease prefers to attack young and vigorous men, who do not as a rule suffer from constipation.

Bad and carious teeth are a very potent predisposing cause of appendicitis. Sir Frederick Treves attributes this to the fact that these people do not masticate their food properly, and that this food in its passage along the alimentary canal tends to ferment and excite catarrhal inflammation. But it appears more probably, in the light of recent work, that carious teeth produce catarrhal inflammation of the whole alimentary canal rather by the condition of oral sepsis to which they give rise. A person whose mouth is in an insanitary condition, and many of whose teeth contain large carious cavities, is constantly swallowing small quantities of micro-organisms and their products, which, when they have overcome the resistance of the stomach by exciting gastritis, are able to extend their pernicious action farther and farther down the alimentary canal.

In a very few cases I have seen appendicitis undoubtedly arise in connection with stercoral ulcers in the cæcum due to the presence of large scybalous masses of fæces.

To sum up the etiology and pathology of appendicitis: The conditions which form the opportunity for the virulent bacillus, when once produced, are likely to persist, so that a person who has once had an attack of appendicitis will tend to go on having them until his appendix is destroyed by inflammation or removed by the surgeon. When these conditions are once produced, they are a source of ever-impending danger. The mine has been prepared in the insidious stage, and it only requires the spark of the virulent bacillus to ignite it.

The Treatment of Appendicitis.

It must be freely admitted that this is a most difficult and complex subject. In the first place, there is probably no disease which is so protean in its form, for in appendicitis we see every degree of severity, from a transient pain up to an illness such as fulminating appendicitis, which falls as it were from the blue, and destroys a strong man in forty-eight hours. Moreover, whilst the majority of cases appear not to relapse within, anyhow, a limited space of time, many others suffer from periodic recurrences at short intervals, making life intolerable. In collecting the facts upon which we should base an intelligent treatment, we also encounter the difficulty that in the specialization of medical and surgical practice cases of appendicitis are so sorted out that various classes of the profession derive their experience from cases exhibiting very different degrees of severity. The general practitioner sees all the slight and doubtful cases, and, unless his practice be a very large one, has only a limited experience of the severe forms, such as fulminating appendicitis and general peritonitis. Of the more severe cases, the hospital physician receives into his wards only those cases which are considered unsuitable for operation, whilst the hospital surgeon gathers his experience from the most fatal cases of this disease, and in this way no doubt gets an exaggerated idea of its general fatality. If we endeavour to derive our facts from published statistics, we cannot but come to the conclusion that these statistics, like other statistics, are quite unreliable. Series of cases published by private individuals would not be published at all unless they were supposed to show a better result than the average, and we must therefore not only be convinced of the honesty of the man who publishes them, but we must know that he has not selected an unusually fortunate series of cases and omitted his less fortunate experience before or after. Fully appreciating and admitting these difficulties, I have endeavoured to glean from the works of all the more important writers on this subject in England and America the fundamental facts which must of necessity guide us in our treatment. These facts are as follows:

1. The attack mortality of all cases of appendicitis, slight or severe, is estimated by Sir Frederick Treves to be about 5 per cent.

2. The attack morality of severe cases derived from the records of a large number of hospitals, cases which were not operated on, is about 20 per cent.

3. There is no doubt that first attacks are much more fatal, and that they are especially fatal in children. Their high mortality is due to the fact that nearly all abscesses and general peritonitis cases arise in the first attack. Any treatment of appendicitis, to be really successful, must therefore aim at forestalling and preventing this high mortality at the first appearance of the disease. A line of treatment which allows all severe first attacks to run their course, and is content to stop relapses by an operation during the quiescent stage, can at best only claim to reduce the general mortality 7 per cent., and passively acquiesces in a mortality of 20 per cent. in these severe cases.

4. About a quarter to a third of all cases relapse within a few years. Dr. Hawkins, of St. Thomas's Hospital, found that one quarter of his cases had relapsed, or did so within the few years which had passed since they had been under his care. Fitz found that two out of five of his cases relapsed.

5. It is estimated that these relapses add about 7 per cent. to the general mortality of all severe unoperated cases. This mortality is of importance when considering the question of an interim operation.

6. Fulminating appendicitis, by which we understand cases of appendicitis which die of general peritonitis or septicæmia within three days of the onset of the attack, forms about 1 per cent. of mild and 2 per cent. of severe cases. It will be noticed how small this percentage is, and therefore it cannot be used, as it has been, as an argument in favour of promiscuous operation, and certainly not for universal operating in cases after the third day.

7. Probably the most important point in the question of operation is the surgeon, whether he is skilled in abdominal work and has had a large and varied experience of appendicitis, or whether, on the other hand, he is inexperienced, and in the nature of things is not likely to have gained any special knowledge of surgical interference in this disease. I have recently had an opportunity of discussing this question with several American surgeons, and they assure me that in the States there is really a terrible mortality arising from the general belief that anybody, however inexperienced, is competent to operate on all cases of appendicitis at all stages of the disease. The nature of the operation is also an important point when considering the amount of skill required by the surgeon. Localized abscesses when obviously pointing can and should be opened by any qualified practitioner. Acute suppurative peritonitis must be dealt with by the man on the spot, for, after all, operation in these cases is a forlorn hope, and gives the patient practically his last chance. The interim operation during a period of quiescence does not in most cases require any special experience other than that possessed by any operating surgeon. Operation during the acute stage is, however, a very serious matter, and requires so much experience, judgment, and technical skill, that it should only be undertaken by those who have had unusual opportunities of dealing with such cases.

8. The interim operation has a mortality in competent hands

of less than 1 per cent. Sir Frederick Treves has published a series of 1,000 cases with a mortality of only 0·2 per cent. But this is not a mortality which can be counted on by every ordinary operating surgeon.

9. The mortality of operation in the first twenty-four hours of first attacks is probably 5 to 10 per cent. in the hands of competent surgeons.

10. Operations on the fourth and fifth day (*i.e.*, seventy-two to ninety-six hours) have a very severe mortality—40 per cent. according to Fowler. When the abdomen is opened, the suppuration is found not yet to be localized, there is no definite collection of pus to be evacuated, and very few indications as to the extent of the procedures which should be carried out. The gut is often distended, and the manipulations and exposure necessary during the operation greatly increase this distension.

11. Operations on localized abscesses give excellent results.

12. Operations on cases of really general suppurative peritonitis give deplorable results. This latter question is complicated by the great variety of meanings given to the term "general peritonitis," but I may say that so great an operator as Sonnenburg of Berlin recently admitted that in thirteen cases of general suppurative peritonitis operated on by him the mortality was 100 per cent., and Mr. Rutherford Morison has said that the results are too horrible to be recorded.

These being the facts which must guide us in our treatment, we will now see how they apply to cases at different stages of the disease. The question of how any case of appendicitis should be treated appears to have been confused by the erroneous supposition that we see all cases from the very beginning, whereas in point of fact they come under our care at very different stages. We shall therefore consider the treatment which we should logically follow out at those stages of the disease which appear to be fundamental.

I. **The First Twenty-four Hours of the First Attack.**—The operative mortality in this stage is about 5 to 10 per cent., according to the skill of the operator, and we must balance this mortality against the 20 per cent. which we have seen is that of severe cases in their first attack, *plus* the 7 per cent. which must be added for the relapses to which unoperated cases will be subjected. (*a*) If a case presents mild symptoms, it is better to wait and watch, but the practitioner must fully recognize that every case of appendicitis, however mild, may at any time develop into one of the most severe. A chart should be kept of pulse and temperature, and these should be observed with the abdominal distension at least every two hours. An enema of soap and water, if not too large, will probably do no harm, and relieve the patient by emptying the colon. The question of whether opium should be given at this stage is a very serious one. Considered theoretically it certainly should not, for it most effectually obscures the signs of the development of the disease, and often leads to the seriousness of the patient's condition being overlooked until it is too late to operate with reasonable hopes of success. On the other hand, I fully recognize how almost impossible it is for the general practitioner to refuse to give his patients relief from their severe pain when he and they are aware that many cases so relieved have developed no serious symptoms afterwards. If at any time severe symptoms supervene, or if the pulse, temperature, or distension, show that the disease is steadily progressing, then the case should be treated as a severe one and operated on at once. (*b*) Where the onset of the attack is sudden and accompanied by severe symptoms, especially by collapse and great pain, an operation should be performed immediately. It is well known in abdominal surgery that the prognosis of ruptured organs, whether they be the stomach, the intestine, or the bladder, is almost entirely a matter of how soon the operation is performed. This matter may be dogmatically summed up as follows: If an operation be performed within twelve hours the patient has a good chance of recovery, but if it be performed between twelve and twenty-four hours his chance of recovery or not are about equal; that between twenty-four and thirty-six hours the operation is still just worth doing, but that after thirty-six hours it scarcely comes within the range of reasonable surgery. It must, however, be remembered, as has been pointed out by my friend Mr. Berry, that the appendix is not a capacious organ containing a large quantity of fluid, and therefore its perforation is only in part comparable to rupture of the stomach, gut, or bladder.

II. **Seventy-two to Ninety-six Hours.**—As we have seen, operation during this period is attended with great risk and a severe mortality (40 per cent.). We must therefore watch all our patients for at least an hour or so. If the pulse, distension, and collapse, and in a less important degree the temperature, are improving, we may continue to wait and watch. But if the pulse, distension, and collapse, are increasing, and the patient's condition is becoming progressively worse, then we should operate, fully recognizing, however, that operation in this stage is a most serious matter, and that our manipulations should be as strictly limited as possible, and carried out with all possible speed.

III. **Sixth to the Fourteenth Day.**—Between the sixth and the fourteenth day purulent collections of fluid in the peritoneum become localized into definite abscesses. We have as a rule a definite tumour; the pulse remains rapid; in spite of the general improvement in the patient's condition, there is often some distension of the intestines, due rather to obstruction of their lumen where they abut upon the abscess than to peritonitis. The temperature also is often somewhat elevated. These abscesses should be opened by suitable incisions as soon as they are diagnosed. But I believe it is a bad practice to search for the appendix in all cases and remove it. This matter will be treated in detail in the next lecture.

IV. **General Suppurative Peritonitis.**—General peritonitis must be operated on as soon as it is diagnosed. We know that general peritonitis is present by the typical facies, the presence of general tenderness and rigidity and the absence of movement over the whole abdomen, by the rapid pulse, which is usually above 130, and by the presence of collapse. A very important sign, to which I think sufficient attention is not paid, is that of the late Mr. Greig Smith. The whole surface of the abdomen is carefully and methodically explored with a stethoscope for a period of at least five minutes, when, if we do not hear the gurgle of any peristaltic movement in the intestines, we may infer that they are paralyzed. The operative mortality of general peritonitis is complicated by the very different meanings given to this term by different surgeons. With some, all cases of appendicitis seem to be regarded as general peritonitis, but, taking the above signs as our gauge of what is meant by general peritonitis, the mortality appears to vary after operation between 75 and 95 per cent. The conservative mortality may be placed at 99 per cent. The 1 per cent. is reserved for those cases which, after refusing operation, get well in spite of all expectation.

V. **After One Definite Attack.**—There is no doubt that after one definite attack of appendicitis the patient should avoid the mortality of 7 per cent. due to recurrent attacks by having his appendix removed in the quiescent period, with a mortality of less than 1 per cent. in competent hands. He is, moreover, spared the pain, discomfort, inconvenience, broken health, and interference with the business of his life, due to these attacks.

VI. **The Treatment of Fistula after Appendicitis.** — These fistulæ are best left alone for at least a year. In the vast majority of cases they heal up within that time. They are not a source of danger, but only of inconvenience. Should they not close within this period, they may be cautiously enlarged and scraped, and, if possible, the fæcal concretion which is very probably present should be removed. If they persist for a greater length of time and show no signs of closing, and more especially if recurrent abscesses occur in connection with them, then the patient may be advised to run the risk of a thorough exploration, with removal of the concretion, if there be one, or what is left of the appendix.

VII. **The Prophylactic Treatment of Recurrent Appendicitis.**— There are many cases in which the symptoms are so slight that there is considerable doubt as to whether they are suffering from appendicular trouble at all. Some surgeons have styled these cases "appendicular colic." It will often occur that for various reasons these cases are not submitted to operation. How, then, are we to treat them and those patients who refuse operation in spite of the strong advice of their practitioner ? In the first place we must see that their teeth are put in thorough order, all stumps removed, and carious teeth extracted or stopped. Deficiencies should be made good by the dentist, and, by means of antiseptic mouth-washes and gargles, the buccal cavity got into as sanitary a condition as possible. Their food should be plain and simple, avoiding such indigestible things as nuts and most vegetables. Their meals should be regular, not eaten in haste, and followed by a suitable period of leisure. If there is any irregularity of the bowels, it should be attended to.

VIII. **The Medical Treatment of an Acute Attack.**—It will have been apparent from my previous remarks that I do not regard appendicitis, at least in its severe forms, as a disease to be treated by the physician. But certain cases refuse surgical treatment or cannot obtain it. These cases are best treated by perfect rest. Their diet should be small in quantity and entirely fluid. Purges should not be administered, but, as has already been said, a small soap enema can scarcely do any harm, and will relieve the patient by emptying the colon.

The question of administration of opium has already been touched upon. If for any reason the idea of operation has been finally abandoned, the cases of slight and intermediate severity certainly appear to get through their attack most comfortably with medium doses of opium. But whilst there is the least prospect of an operation none but the very smallest doses should be administered. Finally, I would impress upon you that the most important point in the medical treatment is to watch the course of the symptoms hour by hour, in order to form an opinion as to when it may be necessary to call in the surgeon.

LECTURE II

APPENDICULAR ABSCESSES*

Gentlemen,— I will begin my lecture to-day by relating to you a case of appendicitis which illustrates many of the points to which I drew attention in my last lecture, and some to which I shall refer to-day. A young man aged twenty-four rushed away one Saturday afternoon last autumn from his office in the City to catch the train from Waterloo Station to Shanklin, in the Isle of Wight. He had no time to get his lunch, and the journey was an extremely cold and inclement one. He arrived in Shanklin exhausted, chilled, and starving. He then ate a very large and apparently indigestible meal. Next morning he was seized with vomiting, pyrexia, and considerable pain in the upper part of the abdomen. On the second day he was jaundiced, and on the third day his temperature was down and he was rapidly improving in condition, when suddenly he was seized with severe pain at the umbilicus and became collapsed. His face, I am informed, was an ashy colour. His medical man, Dr. Cowper of Shanklin, with courage equal to his surgical skill, would not take the responsibility of allowing so acute a case of appendicitis to await the arrival of a surgeon from London, and he accordingly removed the appendix about six hours after the onset of the attack. He found a calculus, and the appendix was flaccid, green, and gangrenous. There were no adhesions, and apparently general peritonitis was present. I saw the patient next day in consultation with Dr. Gilbart Smith. The abdomen was tender all over, but not distended. A considerable quantity of free fluid was located in each flank, and another large collection in the pelvis. Three days later the general peritonitis had subsided from all the upper part of the abdomen. The fluid had been absorbed with the exception of a collection in the pelvis, which formed a soft, fluctuating mass in the recto-vesical pouch. A week later Dr. Cowper asked me to go down to Shanklin, as the collection of fluid in the pelvis had formed a pelvic abscess. On my arrival I found that it had just ruptured into the rectum. The patient made a good recovery. This case illustrates the way in which indiscretions in diet may increase the virulence of the *Bacillus coli* in the alimentary canal, and how this micro-organism attacks the appendix weakened by the presence of a calculus. It is also a good example of sudden gangrene of the appendix leading to fulminating appendicitis, and illustrates how these cases can only be saved by immediate operation. The free fluid mentioned in the above case is antibacterial in action, and preserves the peritoneum from invasion by the micro-organism. The pelvic abscess was due to the whole of this fluid not being absorbed, and the rupture into the rectum, I will point out later on, is the most favourable termination of this form of appendix abscess.

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* Delivered at the Polyclinic on February 27, 1903.

Appendix abscesses form in two ways: Either adhesions are formed before the suppurative process extends from the appendix, or suppuration breaks into an open peritoneal cavity, and leads to a great outpouring of lymph into the peritoneal pouches, some at least of which may subsequently form abscesses. We will take these in turn.

When suppuration starts inside the appendix, its peritoneal covering becomes inflamed, lymph is deposited on it, and the neighbouring coils of small intestine become matted together around it. If, after this has occurred, the suppuration spreads through the walls of the appendix, it finds itself shut in and circumscribed by a barrier of adherent coils of gut. In the great majority of such cases the appendix is actually perforated, and its interior in continuity with the abscess formed. This is, however, not necessarily the case, and the micro-organisms may pass through the wall of the appendix and form an abscess around it without any actual perforation. It is these latter cases which supply the small minority of appendix abscesses which relapse after they have been drained and healed.

In the second class of appendix abscess, either because the appendix ruptures, or the micro-organism is of such virulence that it rapidly passes through the wall of the appendix, there is no time for the formation of any adhesions, and the micro-organism enters an open peritoneal cavity. The first measure of defence is the outpouring of a large quantity of serum into the peritoneal cavity, which stands in ponds in the peritoneal pouches, to whose anatomy we must refer immediately. This serum is antibacterial in action, and serves to arrest, or at least to delay, the invasion of the entire cavity by the micro-organism. Should it be successful in this endeavour, the area of infection is circumscribed by adhesions, and the serum is absorbed from the rest of the peritoneal cavity. Should the infection, however, be too virulent, leucocytes pass in vast number into these ponds and add their antibacterial action to that of the serum, and in this way convert them into cavities filled with pus. The leucocytes, moreover, block up the stomata of the serous membrane, and, as masses of lymph, wall in and circumscribe the septic area, so that even the toxin which the micro-organism succeeds in forming in the abscess is not absorbed into the general system to poison it. We must now turn for a moment to the clinical side of the question, and consider *the signs and symptoms which will inform us in any case of appendicitis when suppuration has commenced.*

It has been well said by Dr. Hawkins of St. Thomas's Hospital that no man can foretell in the first forty-eight hours of an attack of appendicitis whether it will terminate in suppuration or not. The mildest attack, attended by a low temperature and only a slight pain or two, may yet end in a large abscess, whilst another case, in which there were severe symptoms and high pyrexia, may yet resolve in three or four days without suppuration having occurred. It is, however, certain that severe attacks more often terminate in pus than slight ones. To quote again from Dr. Hawkins, the indication that suppuration has occurred is not the appearance of any new sign, but a continuance or increase of symptoms which should be abating on the fifth or sixth day. We should strongly suspect that pus has commenced to form if after the fifth day of appendicitis the temperature has not reached normal or is remaining high, or after having come down has risen again. The pulse is also a valuable indication if it remains rapid, but I have often seen a well-circumscribed abscess with a pulse-rate of not more than eighty-four. Diarrhœa is a valuable sign; but perhaps the creamy tongue, which has not commenced to clean, and the pale, pasty, and muddy complexion of the patient, are the most suspicious symptoms. This aspect of the patient is very striking in some cases, and might almost be called a purulent complexion. After the abscess has been efficiently opened and drained, it almost immediately changes to a clear and rosy countenance. We may, perhaps, connect this change of appearance with the great alteration in the number of leucocytes circulating in the blood which accompanies it, for whilst the abscess is undrained there are from 23,000 to 30,000 leucocytes per cubic millimetre in the blood, and with the draining of the abscess this number falls to 8,000 or 10,000. As we saw in the first lecture, abscess is much more common in the first attack. Of the 27 cases in which I made a special note of the attack, the abscess formed in 20 during the first attack, in 4 during the second, and in only 3 afterwards; and in 29 cases in which I noted the day on which the abscess was discovered, in 23 it was after the sixth day, and in only 6 cases were abscesses found on the fourth and fifth day, and in these considerable general peritonitis was present.

When on these general grounds we suspect that pus is present, we must discover and watch the tumour formed by the inflamed appendix with the greatest attention. It is very bad practice to await the appearance of redness, œdema, or fluctuation; one might as well await their appearance in empyemata. For many abscesses will have ruptured into the general peritoneal cavity before they produce these signs in the skin. We therefore diagnose the existence of an abscess if the lump in the abdomen continues unchanged, or especially if it increases in size after the fifth day.

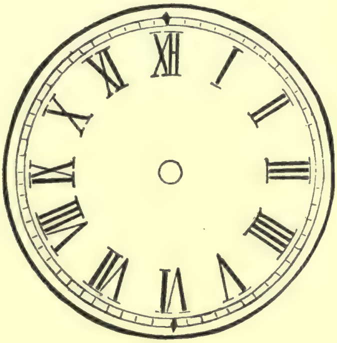
The position of appendix abscesses is determined partly by the position in which the appendix lies, and partly by the peritoneal fossæ in its neighbourhood which the effusion of serum may occupy. The appendix may either lie inside or outside the cæcum and colon, and if it lies inside the cæcum its tail may be either in front or behind the mesentery of the small intestine. We may therefore classify the positions of the appendix as follows:

A. External.		B. Internal.	
1. Lumbar - - -	= 4 cases.	1. Iliac - - - - - Behind mesentery.	= 10 cases.
2. Subhepatic - -	= 4 ,,	2. Promontory - - In front mesentery.	= 2 ,,
3. Subphrenic - -	= 4 ,,	3. Pelvic - - - Behind mesentery.	= 14 ,,
4. Retrocæcal - -	= 4 ,,	4. Poupart - - - Neither.	= 2 ,,

I have noted the position of the abscess in 44 cases distributed as above.

The clock mnemonic (see p. 307) is a useful means of remembering these positions of the appendix, with their direction and significance. The appendix is compared to the hand of a clock, and is supposed to rotate around its attachment to the cæcum.

The first of these, the iliac position, is the commonest. The appendix, coming off from the posterior and inner part of the cæcum, runs upwards and inwards behind the last piece of the ileum, and is shut in anteriorly by the end of the mesentery. An abscess formed in this situation is therefore well cut off from the general peritoneal cavity. As it extends the pus comes forward in the angle between the ileum and the cæcum, and reaches the anterior abdominal wall in this situation. Clinically we find the tumour situated under McBurney's point, with an interval between it and the anterior superior spine and Poupart's ligament.



I o'clock.—Sacro-iliac synchondrosis—usual site behind mesentery.
II o'clock.—Promontory—among coils of small gut—in front of mesentery—a fulminating position.
III o'clock.—Crosses iliac vessels—hangs into pelvis—felt in recto-vesical pouch—behind mesentery.
VI o'clock.—Middle of Poupart—fœtal form—dangerous—points at times through rings.
XI o'clock.—External—in furrow—felt in loin—spread subphrenic, and rarely small sac.
XII o'clock.—Extraperitoneal—retrocæcal—psoas myositis—perinephritic—subphrenic—bladder—pelvis of kidney.

In the second of the internal positions the appendix passes from its origin upwards and inwards toward the promontory, but passes in front of the mesentery and behind the omentum. Here it lies in the centre of the abdomen and among all the coils of small intestine, with no part of the abdominal wall anywhere near it. I consider this position to be a most dangerous one, and in the only two cases in which I have definitely located the appendix in this situation it has led to fulminating peritonitis.

In its third position the appendix passes directly inwards across the iliac vessels and beneath the mesentery, and hangs dependent into the recto-vesical pouch in the male, or Douglas's pouch in the female. We shall consider this pelvic abscess in detail later. But it may be said at once that the abscess formed by an appendix hanging into the pelvis can only be reached by a finger in the rectum or vagina.

In its fourth situation the appendix passes directly downwards, and lies upon Poupart's ligament in the inguinal furrow. This always occurs in the fœtal form in which the appendix arises from the extremity of the cæcum. It is in my opinion a dangerous form of appendix abscess, for, lying as it does at the edge of the mesentery, the pus may either pass in front or behind that structure; and should it escape between the omentum and the mesentery, it will reach the central part of the abdomen occupied by the numerous coils of small intestine which are most sensitive to infection and produce the greatest shock. This abscess can easily be felt as an indurated mass lying above the outer half of Poupart's ligament. It sometimes escapes by the inguinal canal and the external ring.

In Fig. 45 are illustrated the peritoneal pouches, which will be invaded by an abscess originating from an appendix which lies between the ascending colon and the right loin. Such an appendix may be long enough to reach the lower border of the liver, and lie either outside it beneath the diaphragm, or, passing below its inferior surface, may touch the gall-bladder. But it is not necessary for an appendix to actually reach these situations in order to produce abscesses there. In this figure, No. 1 represents a lumbar abscess. As you will see, it is shut in between the ascending colon and the loin, and the twisted mesentery of the appendix serves to prevent pus passing downwards. Clinically it produces a tender, resistant tumour, passing back along the iliac crest from the upper part of the iliac fossa, and extending up the loin both laterally and behind. Indeed, the best sign of an appendix in this situation is the extreme tenderness on deep palpation at the outer border of the erector spinæ. If the pus extends up to the lower border of the liver, it readily passes below it to the left, being bounded below by the transverse colon, above by the liver, and behind covering the upper two-thirds of the right kidney (Fig. 45). Such an abscess produces a large area of dulness and induration on the right side of the abdomen in front, continuing downwards from the right costal margin and the lower border of the liver. The peritoneal fossa in which this abscess forms is the same one into which the bile tends to drain after operations on the gall-bladder and bile-ducts, and Mr. Rutherford Morison of Newcastle has fully described its anatomy in this connection, and has recommended that it should be drained in these biliary cases from the loin. In

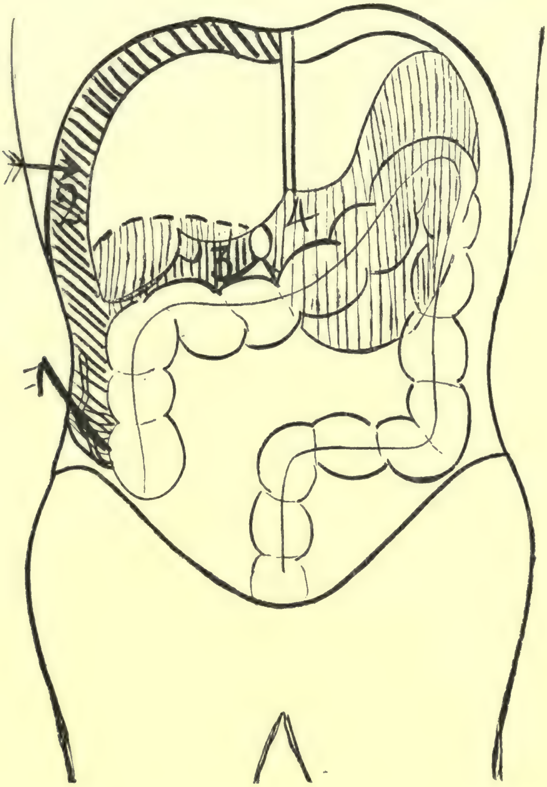


Fig. 45.—Illustrates the Spread of Pus along the Chain of Right Peritoneal Ponds from an External Appendix Abscess.

1, The appendix in external position lying in a lumbar abscess; 2, subphrenic intraperitoneal abscess between liver and diaphragm, and limited to the left by the falciform ligament; 3, subhepatic abscess between the lower surface of liver and the upper end of right kidney; 4, small sac of peritoneum very rarely invaded through the foramen of Winslow from 3. The oblique loin incision is represented; through it tubes may be passed into 2 and 3. When 2 is shut off and circumscribed, it must be opened across the pleura in the line of the arrow.

a few rare cases the suppuration has extended still farther to the left from this subhepatic fossa by passing through the foramen of Winslow and distending the sac of the small omentum, as is shown in Fig. 45 (4).

To turn back to the point at which the pus reached the lower border of the liver, it may as well as passing below the liver pass between that organ and the diaphragm, and form a large subphrenic abscess in the situation numbered 2 in Fig. 45.

We shall refer to the clinical signs by which this subphrenic abscess may be recognized when we come to deal with these abscesses in detail. The last position of the appendix is one in which it passes directly up behind the cæcum and ascending colon, and lies apparently outside the peritoneal cavity altogether, in relation with the psoas muscle, the right kidney, and the cellular tissue around these structures. But this abscess also will be dealt with in detail later.

I will now describe to you *the principles which should guide us in opening any of these abscesses*, with the exception of the pelvic, subphrenic, and retrocæcal abscesses, which will require special treatment. The situation and limits of the mass formed by the abscess should be most carefully defined, and we should then so plan our incision through the anterior abdominal wall that it may be as far removed as possible from the general peritoneal cavity. We should, indeed, approach the abscess from the outer side, in order that as little risk as possible may be run of infecting the general peritoneum by allowing the pus to pour into it as we release it from the abscess. No formal incision is suitable in every case, and you will see by the diagrams I show you the deplorable results of making orthodox incisions where they are not applicable to the abscess which is being opened. For these figures I am indebted to Mr. James Berry. He drew them from actual post-mortems made by him at St. Bartholomew's Hospital. The adhesions formed by the abscess to the anterior abdominal wall are marked in each case, and you will see that in scarcely any instance was the incision made within this region. In nearly every case the surgeon incised either in the middle line below the umbilicus and entered the abscess cavity from the general peritoneal cavity, infecting it by so doing, or his incision was the orthodox oblique one opposite the anterior superior spine, and in a similar way the pus was allowed to run from the abscess into the open peritoneal cavity.

As layer after layer of the abdominal wall is cut through, we must notice the presence or absence of œdema between these layers as we approach the peritoneum. If well-marked œdema is present, we can be certain that the abscess has formed adhesions to the abdominal wall, and whilst there is little risk of infecting the general peritoneal cavity, we must avoid wounding the adherent coils of gut. Should open peritoneum be encountered, we must pack it off with a barrier of gauze all round before proceeding any farther. For this purpose it is usual to employ broad iodoform gauze with a woven edge, and to systematically pack it in around the incision, starting at one end from the point where the abscess wall is adherent to the abdominal wall, and working systematically round the wound to the point on the other side where it is similarly adherent, so that there may be no crack or crevice leading from our wound into the peritoneal cavity which is not blocked by the gauze barrier. The effectiveness of this method in protecting the general peritoneal cavity from infection has often filled me with astonishment, when I have seen foul and stinking pus pouring from a large appendix abscess and beating against the narrow barrier of iodoform gauze which I have placed to prevent it flowing into the peritoneum. It seems that this gauze acts in several ways: mechanically it serves as a dam, and makes it easier for the pus to flow from the wound than to percolate through the gauze; then it appears that it acts as an irritant upon the coils of gut upon its farther side, and makes these adherent to one another before the micro-organisms have overcome the obstacle it offers to their advance. It is possible, moreover, that it acts as a filter, straining off the micro-organisms themselves, but permitting their toxins and poisons to soak through its substance and excite a large formation of lymph, which effectually bars the progress of the bacteria themselves when they reach it. When the gauze barrier has been prepared, the index-finger is employed to break down the adhesions between one coil of gut and the next, and to open a way into the abscess cavity. This aperture, again, should always be made on the outer side, and as far as possible from the central region of the abdomen. There has been much dispute as to whether or not the appendix should be removed when an abscess is opened. It is claimed that some cases of appendix abscess relapse even after the abscess has been drained and healed. But these cases of relapse are very few. In Dr. Hawkins's cases one relapsed in twenty-three, and in a great number of cases collected by Coley the relapses were estimated at 20 per cent. In forty-two cases of appendix abscess of which I have notes, only one case has had recurrence, but these cases have occurred only in the last three years. We must remember, also, that the drainage of well-localized appendix abscesses is attended by a mortality of not more than about 5 per cent., but that if the appendix is sought for and removed in every case it will necessitate a large incision, with its greater risk of ventral hernia, extensive breaking down of the adhesions, which must in many cases lead to general peritonitis and a very much prolonged operation, which must take a further toll in consequence of the shock it produces. Supposing that even 20 per cent. relapsed, these may be operated on in the quiescent period with the 1 per cent. mortality to which we have so often referred. When the abscess has been opened, a thorough but gentle search should be made for fæcal concretions or calculi. For if they are left in the abscess cavity they will lead to the formation of a fistula, and unless they are ultimately disintegrated and discharged the abscess will recur. I believe that the largest drainage-tubes should always be employed for these abscesses, and I prefer them ¾ inch in diameter. After the second day a full-sized soft catheter attached to a syringe may be inserted to the bottom of this large drainage-tube, and the cavity of the abscess be thoroughly well washed out with 1 in 80 carbolic lotion. This washing is continued until the lotion comes back clear, and the abscess cavity is then sucked dry by reversing the action of the syringe. These big tubes should not be taken out to be cleaned during the first week, for it will be found very difficult and painful to replace them, more especially as, when they reach into the pelvis, they are six or seven inches long. They should, however, be turned round each day, in order that the lateral openings may not become blocked.

Pelvic Abscesses.

I wish to especially draw your attention to pelvic abscess in appendicitis, because in the first place it is far more common than seems to be supposed; and, secondly, of all appendix abscesses it is the most insidious, with the exception, perhaps, of the subphrenic abscess, to which I shall refer later. It is a deplorable thing, when a patient has safely passed through all the dangers attending the first week of an acute attack of appendicitis, and appears wellnigh out of danger, that he should then succumb to a pelvic abscess which has lurked in the pelvis and developed until it has finally involved the whole peritoneal cavity and led to the patient's death at this late stage of the disease. The reason that pelvic abscess is so often overlooked is because it can only be diagnosed by examination from the rectum or vagina. It is therefore an absolute axiom of appendix surgery that in every case of appendix abscess the rectum must be examined. And it is a useful aphorism that if in a case supposed to be appendicitis the appendix cannot be located in the abdomen it will be found in the rectum.

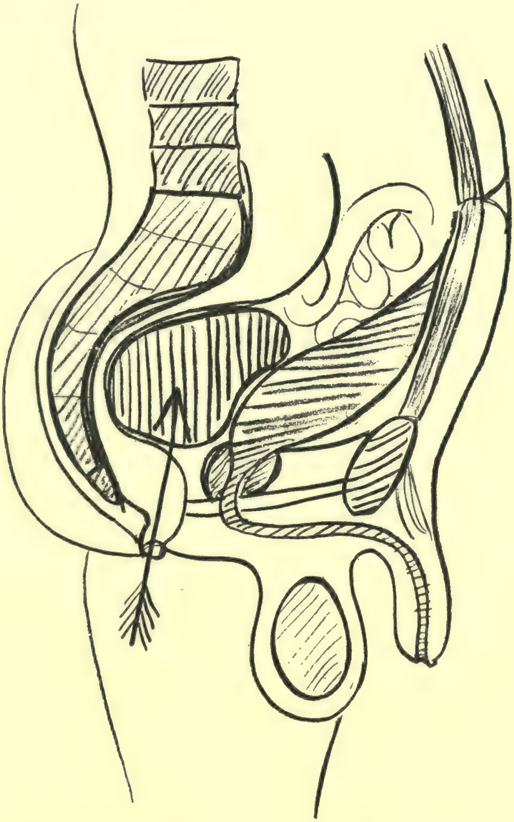


FIG. 46.—A PELVIC APPENDIX ABSCESS IN THE MALE IN THE RECTO-VESICAL POUCH.

It is easily felt from the rectum just above the prostate, as a more or less fluctuating tender mass. It is best opened at this point, as the arrow indicates. It compresses the rectum and dilates the entire colon above it. It presses on the bladder, and more often than in the female leads to frequency or retention. Always empty the bladder with a catheter before opening such an abscess from the rectum. Matted coils of gut roof it in above.

The abdominal condition which should arouse our suspicion that an abscess is deeply seated in the pelvis is as follows: The abdomen is generally distended and tumid, but it is not sufficiently tender or rigid for us to attribute this distension to peritonitis.

This distension is indeed due to obstruction of the rectum in the pelvis by the abscess there (*vide* Figs. 46 and 47), and as a consequence the entire colon, and in advanced cases the small intestine, becomes dilated. No definite lump can be found in the iliac fossa or elsewhere. But in advanced cases there is an obscure resistance above the pubes, due to the dome formed by matted coils of gut above the abscess cavity. This obscure resistance is resonant on percussion. In nearly every instance I have, however, found a tender spot over McBurney's point. In many cases the abscess, lying as it does between the rectum and bladder, produces symptoms referable to irritation of these organs. There is diarrhœa and tenesmus, with the passage of frequent motions containing much slime, and the urine may be either frequently voided, or in rarer cases actually retained. In one case the signs and symptoms were much complicated by retention, and a bladder distended with 24 ounces of urine. When this had been withdrawn by a catheter, a further large fluctuating mass was found behind it, which proved to be a pelvic appendix abscess. In a case exhibiting such signs, if we examine by the rectum, we find the condition shown in Fig. 46. Situated above the prostate and in front of the rectum is a large, tender, sometimes hard and sometimes fluctuating mass, frequently the size of an orange. The mucous membrane of the rectum over it is often swollen, œdematous, and secreting the slime to which we have already referred. The arrow in the diagram indicates not only the point at which the examining finger will impinge upon the abscess, but also the point at which it is best opened, as we shall describe later. We also see in the figure how the rectum is flattened against the sacrum behind, and how in front the abscess compresses the neck of the bladder, whilst above is indicated the matted coils of gut which shut the abscess in and separate it from the anterior abdominal wall. In the female (Fig. 47) the abscess can in addition be palpated in the posterior fornix of the vagina, where Douglas's pouch will be found distended and bulging; and, as I shall point out later, this is the point of election from which to open such an abscess. It will be readily understood that an appendix hanging down into Douglas's pouch will be in close relation to the right ovary and Fallopian tube, and in its early stages, when adherent to the ovary, it may be very difficult to tell whether the pain is due to ovaritis or salpingitis, or to an inflamed appendix adherent to these structures. In some cases it is even difficult to be certain in which of these organs the mischief arose, after the abscess has been opened into the vagina. But the character of the pus, which is usually characteristically foul in appendicitis, will in most cases correctly indicate its origin.

I have during the last three years closely investigated fourteen cases of pelvic appendix abscess which had reached different stages of development, from a localized collection of pus in the pelvis up to general peritonitis which led to death; and I have been able from this investigation to trace the course by which the abscess spreads during the latent or insidious period, and this

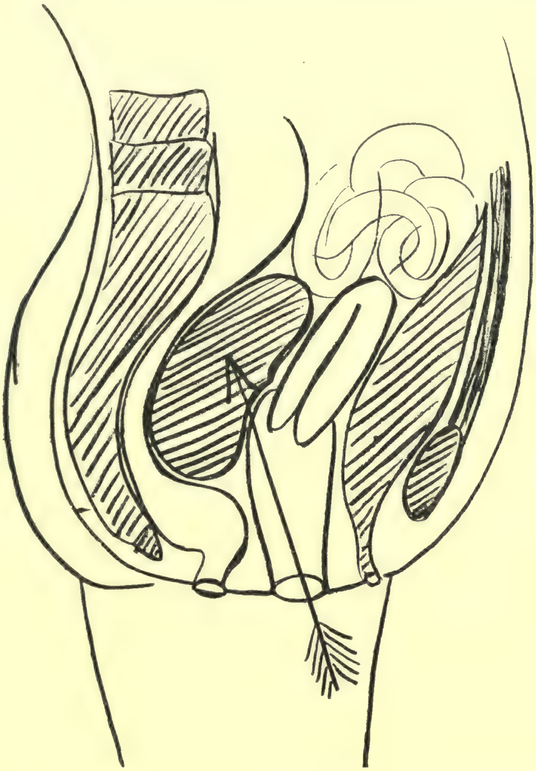


FIG. 47.—PELVIC APPENDIX ABSCESS IN THE FEMALE.

It is felt from the rectum in Douglas's pouch, and obstructs the rectum, distending the whole colon. It is felt from the vagina in the posterior fornix. The arrow shows how and where it should be opened. It compresses the bladder in front, causing painful and frequent micturition, and, very rarely, retention. The dome of matted coils of gut above shut off the general peritoneal cavity.

course is shown, as it were, in a map in Fig. 48. In many cases pelvic abscesses perforate the wall of the rectum and discharge themselves as a sudden fluid motion. I have in two such cases been able to identify with my finger the point of perforation. This termination is a very favourable one, for in a fortnight all discharge has ceased, and nothing remains but some induration in the recto-vesical pouch. In other cases, however, the abscess, starting from its point of origin in the pelvis, gradually expands and lifts the firm dome of adherent coils of gut as though it were the lid of a pot, and the pus then escapes round the edges behind into the right or left iliac fossa (*vide* Fig. 48). Since the abscess at its start is behind the mesentery, as it spreads it keeps behind the small intestine, and these pushed forward against the abdominal wall serve to conceal it as it creeps from the iliac fossæ on either side to the loin, and from the right loin below the liver and diaphragm, and on the left around the spleen. In a recent case in a boy I found large collections of pus in all these situations, and yet the temperature was not more raised than 100° F., the pulse was below 100 per minute, and the abdomen exhibited nothing except diffuse distension. The deep posterior situation of the abscess as it spreads is very fortunate in one way, for the pus in its entire course skirts round the edge of the mass of intestines, and does little harm where it lies shut in against the posterior abdominal wall; but the very effectiveness of this isolation makes this stage of development insidious, and leads to its being overlooked unless it is suspected. There are two other collections of pus which may extend from those I have mentioned. The subphrenic abscesses may infect the pleura through the diaphragm, and lead to local or general empyemata at the bases of the lungs. In my wards at the London Hospital at the present time is a young fellow in whom a pelvic abscess tracked up the left loin and formed a left subphrenic abscess. Both these collections of pus were emptied, but two days later he coughed up a large quantity of stinking pus and gas, apparently from a localized empyema at the left base; and this last collection is being evacuated so well by these natural means that I do not propose to resect a rib—at least at present. At post-mortems on two of these pelvic cases which came into the hospital with general peritonitis due to the final development of a pelvic abscess, in one case, fourteen days after the onset of the attack, I found a further collection of pus (*vide* Fig. 48), which was situated between the descending colon and the spine in the hollow of the left loin, roofed in by the posterior surface of the mesentery; and I have not been able to determine whether this is the usual route

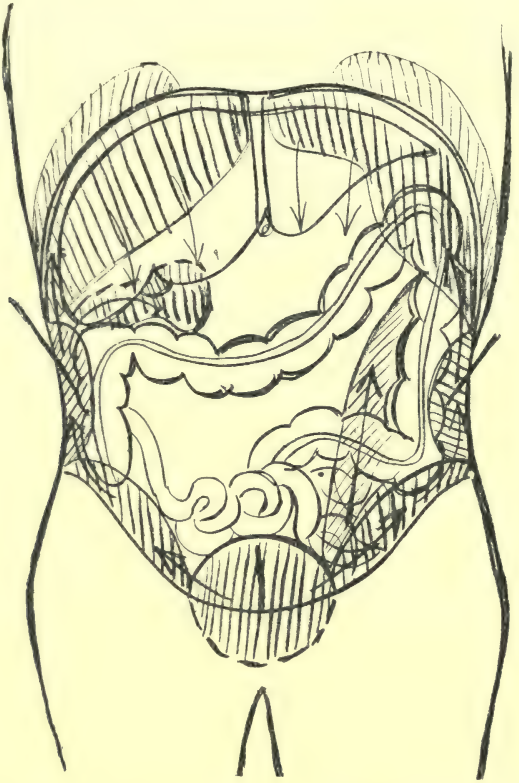


FIG. 48.—ILLUSTRATES THE SPREAD OF A PELVIC APPENDIX ABSCESS ALONG THE RIGHT AND LEFT CHAINS OF PERITONEAL PONDS.

The abscess is at first deeply localized in the pelvis (see Figs. 46 and 47). It then lifts the dome of matted gut and escapes into one or both iliac fossæ. On the right it then spreads along the pouches already illustrated in Fig. 45, to the right subphrenic space. On the left the pus similarly reaches the subphrenic space around the spleen. In some cases it passes between the descending colon and the loin; in others, between the descending colon and the spine, lying on the front of the kidney, and shut in by the back of the mesentery. Up to this stage all these abscesses may be drained by the incisions indicated in the figure—one median one, and two oblique ones far back in the loin. Finally the subphrenic abscesses come down the front of the liver and invade the central dangerous area of the abdomen, or spread through the diaphragm, producing empyemata or pericarditis.

by which pus escaping from a pelvic abscess passes up the left loin and reaches the left subphrenic space, or whether, as on the right side, it tracks up outside the colon. In its final stage of development, the subphrenic abscesses right and left rupture from above, and the pus passes down towards the central region of the abdomen. Then acute symptoms of general peritonitis at once supervene, and this is, unfortunately, often the first point at which the doctor becomes aware that the case is not progressing as it should. Up to the point at which this occurs the patient is still in good condition, and may be saved by suitable incisions, so planned as to drain all these collections of pus. These incisions, which are marked on the diagram, are first a median one just above the pubes. After gauze has been packed in from one Poupart ligament to the other in the form of a rainbow, the index-finger is steadily bored down the back of the bladder, perforating the dome of adherent coils of gut, until it slips into the cavity of the abscess. A large drainage-tube about 6 inches long is then passed down to the bottom of the pelvis. Two lateral incisions are also necessary, one in each loin. These are placed as far back as possible, and through the right one tubes are passed below the liver, under the diaphragm, and down the loin to the iliac fossa. Through a similar incision on the left the abscess around the spleen is drained above, and another tube is passed down into the left iliac fossa. A small boy of twelve, to whom I have already referred, was treated by these incisions two months ago. A pelvic, two iliac, two lumbar, and two subphrenic abscesses were opened, and he is now convalescing at the seaside with all his sinuses healed.

The Treatment of a Pelvic Abscess.—It is always better to drain a localized pelvic abscess from the rectum or vagina. By either of these methods the patient is well in a fortnight, and never in the hospital more than three weeks. Moreover, they run no risk of ventral hernia. On the other hand, if they are drained by a suprapubic incision, the wound takes six weeks at least to heal, and often much longer. It must, however, be admitted that the cases which I drained from the abdomen were more severe and extensive cases, or I should not have adopted this method. If, however, the abscess has already spread to the iliac fossæ and the loins, it is not enough to open the pelvic abscess. How, then, can we tell whether a pelvic abscess is still localized as shown in Figs. 46 and 47, or whether it has already started on the development shown in Fig. 48?

We can, guided by a knowledge of the course it will take, palpate the iliac fossæ and the loins, but, as I have already pointed out, the pus is very deeply situated, and is covered by all the coils of small intestine. Some fulness can, however, usually be discovered in these situations. A more valuable sign is marked tenderness on deep pressure at the outer edge of the erector spinæ in the loin behind. The best indication of all, however, is gained by palpating the abscess cavity with a finger in the rectum. If it is hard and tense, the abscess is still shut into the pelvis. Directly the pus escapes over the brim of the pelvis, the abscess as felt from the rectum becomes soft and flaccid, and can with difficulty be distinguished from a distended bladder or dilated coils of gut.

Should we then have decided that the abscess is still limited to the pelvis, and the patient is a male, the abscess should be opened into the rectum (*vide* Fig. 46).

The anus is first dilated, the sphincter paralyzed, and the bladder is emptied by a catheter. The rectum is well douched. The index-finger searches for a soft spot on the tense mass above the prostate, and a large exploring needle on a syringe is plunged into it. If pus is found, a sharp-pointed director follows the needle, which is then withdrawn, and sinus forceps are passed up the groove of the director, and by opening these a hole is torn sufficiently large to admit the index-finger. A large tube is then inserted, and cut short enough to rest on the upper surface of the sphincter. Rectal washes are given twice a day. The tube can be discarded after three or four days, and the patient is allowed up as soon as the discharge of pus ceases, which is usually in fourteen days.

Should the patient be a female, it is, I think, better to utilize the vagina for drainage (*vide* Fig. 47). With curved scissors the mucous membrane and the vaginal wall are cut through in the middle line immediately behind the cervix. A director is passed into the pus cavity, and the procedure is then exactly as in the male. The results are just as good.

When I commenced to open pelvic abscesses, I had a great antipathy to doing so from the rectum, for this appeared to me a dirty procedure, and one likely to infect the abscess cavity. At this time I heard of a case drained by Mr. Goodsall through the ischio-rectal fossa, and I adopted this method in two cases. An inch incision is made into the ischio-rectal fossa by the side of the anus, and the index-finger bores its way through the fat to the top of that cavity. A sharp director is then passed up the finger and impinged against the levator ani muscle which separates it from the abscess. The other index-finger in the rectum serves as a guide. The director is then bored through the levator ani and recto-vescial fascia into the recto-vesical fossa, entering it from the side. Sinus forceps are passed in and the opening dilated as much as necessary. On the third occasion on which I followed this procedure, the abscess burst into the rectum as the director was being bored through the dense tissues—a procedure which requires considerable force—and this patient did perfectly well. Soon afterwards the abscess ruptured into the rectum, whilst another case was being examined on the table prior to making the ischio-rectal incision. This case also did well. I then abandoned the ischio-rectal route, as it presented no advantages over the rectal method, and had, indeed, several disadvantages. The sinus took some week or so longer to heal, and a colleague who employed the method encountered fairly severe hæmorrhage. Finally, it appeared to me that, if the force required to perforate the levator ani and recto-vesical fascia were enough to burst the abscess into the rectum in one case, in another it might rupture t into the general peritoneal cavity.

Should the signs which I have already enumerated, of fulness in the iliac fossæ, tenderness in the loins, and a flaccid mass felt by the rectum, indicate that the pelvic abscess has already spread to the iliac fossæ, or even farther, then the abscess must be opened in the middle line above the pubes. Before the abscess is opened or the adhesions disturbed, its limits may be cautiously explored from this median incision and the necessity of lateral loin incisions determined on. When this point has been decided, the rainbow packing of gauze is inserted from one of Poupart's ligaments to the other, and the abscess opened as already described.

On one or two occasions when I had found pus in one loin and drained it, and proceeded to incise the other, I found so little pus there that it was uncertain whether it had reached the subphrenic or subhepatic spaces.

I nevertheless passed the exploring finger into these pouches, and as it was withdrawn as a rule a gush of serum more or less turbid followed it. But after these pouches have been thus exposed to contamination, a tube should always be passed into them whether they were found to contain pus or not.

Sometimes, whilst opening an obvious collection of pus in the right iliac fossa, one suspects from its distribution or from a previous rectal examination that there is a secondary collection in the pelvis, as was the case in the patient whose history I related to you at the beginning of this lecture. The index-finger should then be slipped across the iliac fossa until it passes the iliac vessels and drops into the pelvis, when serum or pus will gush out along it. A tube should then be slid down into the bottom of the pelvis. This tube will require to be 6 or 7 inches long.

I have already described the method of draining an advanced case of combined pelvic, iliac, lumbar, and subphrenic abscesses. The incisions may be seen marked on Fig. 48.

Appendix Subphrenic Abscesses.

The history of these abscesses is nearly always as follows: An external appendix abscess has been opened by an incision in the right side running a greater or less distance back into the loin (*vide* Fig. 45). Although apparently the abscess is well drained, the patient does not do well. He displays those constitutional signs of appendix abscess to which we referred at the beginning of this lecture. His temperature is still raised, his pulse does not come down to normal, his tongue is creamy, his complexion pale, pasty, and muddy. Perhaps he has five or six motions a day. The abdominal examination reveals no further collection of pus. The pelvis is clear. Where, then, is the pus? Under these circumstances we must repeatedly examine the base of his right lung for signs of pus hidden beneath the dome of the diaphragm.

If the original abscess was a pelvic one, the bases of both lungs must be most carefully watched (*vide* Fig. 48). If there be an abscess, the line of liver or splenic dulness will be found to slowly rise towards the angle of the scapula. A sudden increase of dulness in this region indicates that the pleura, in anticipation of invasion from below the diaphragm, has poured out a copious flood of antibacterial serum. The stethoscope will in some cases reveal the tubular breathing of compressed lung, in others a harsh pleural rub, or, where pleural effusion has occurred, the absence of breath-sounds. As soon as the signs justify it, a large exploring needle on a syringe should be inserted. I usually do this in the line of the angle of the scapula, and between the tenth and eleventh ribs; and if no pus is discovered at a depth of 3 inches, I explore in a similar way the ninth, eighth, and seventh spaces. When the needle enters the liver dark venous blood is withdrawn. When the blood is withdrawn from the lung it is bright and scarlet, and often contains air-bubbles, or even foams.

Not only in this way can one tell that the point of the needle is through the diaphragm, but if this is the case at each inspiration the syringe is tilted upwards as the needle is carried down by the diaphragm. It is possible to withdraw pus from below the diaphragm and clear serum from above it in certain cases.

I have here a patient who exhibited this sign. He had an external appendix abscess which was opened and drained through an oblique incision in the right loin, the scar of which you can see. Subsequently he exhibited the signs and symptoms of a further collection of pus, the situation of which was indicated by a rapid rise in dulness from his liver to his interscapular region on the right side.

Under an anæsthetic a needle was inserted into his ninth intercostal space in the line of the angle of his scapula; a syringeful of foul pus was immediately withdrawn. Two inches of his ninth rib were resected subperiosteally. Curved needles were then passed so as to suture his diaphragm to his intercostal muscles, and in this way shut off his pleural cavity before it was opened. An incision was then made through the diaphragm, and about ½ pint of foul pus and gas released from under its dome. The question then arose as to what was the cause of the dulness so high in the thorax; another needle was accordingly inserted between the fifth and sixth ribs in the interscapular region, and clear serum was withdrawn. Dr. Bulloch, bacteriologist to the hospital, reported on both specimens. The pus contained *Bacillus coli communis* and *streptococci*. The serum was sterile.

In this other patient the history was almost identical, but the subphrenic abscess was sooner discovered, and was smaller and more restricted to the top of the dome of the diaphragm. It was accordingly the eighth space in which the needle struck pus, and the eighth rib was resected.

In yet another case in a young Jewess, when the ninth rib was resected, the diaphragmatic and parietal pleura were found adherent to one another by lymph so that they could not be identified. I unfortunately omitted to insert sutures fixing the diaphragm to the intercostals. For two days she did well; then, as the abscess beneath it emptied, the diaphragm descended, and in the middle of the night it tore away from its attachment to the chest wall. Air freely entered the right pleura, and the lung collapsed. The respirations rose to 54, and the pulse to 150. However, she rallied, and the lung re-expanded in due course.

Ultimately she made a good recovery. These sutures should therefore never be omitted, whether the diaphragm is adherent to the chest wall at the time of operation or not.

As I have already stated, if a collection of serum or pus is suspected at the time an appendix abscess is opened in the loin, the index-finger is slipped up to the edge of the liver and is inserted below it, and between it and the diaphragm. Tubes are then introduced whether pus is found or not.

Retrocæcal Appendix Abscess.

An appendix lying behind the cæcum and ascending colon, and outside the peritoneal cavity altogether, is anatomically unknown.

Pathologically, however, we frequently find an appendix in this situation, and from its extraperitoneal position it may produce most anomalous signs.

Fig. 49 shows you the two ways in which this condition may be brought about.

More commonly the appendix was originally an external one (Fig. 49). It was well tucked down into an unusually deep groove between the ascending colon and the loin. When it became inflamed, dense adhesions were formed outside it between the ascending colon and the parietal peritoneum, and as these became converted into fibrous tissue the appendix came to lie directly behind the colon, and practically outside the peritoneal cavity altogether. I have seen several stages of this process of exclusion from the peritoneal cavity.

What I believe to be a rarer origin of a retrocæcal appendix is when it is placed in an unusually deep retrocæcal pouch of peritoneum (Fig. 49, *c*).

In whichever manner it reaches this extraperitoneal position, it lies in consequence of it in very close relation with the right psoas muscle, the right kidney, and ureter, and the cellular tissues around these structures. In some cases the signs when such an appendix becomes inflamed, and especially when it suppurates, are chiefly referable to the psoas muscle.

In such a case which I saw under the care of a colleague, the hip was flexed at a right angle, and the boy would allow it on no account to be extended. His temperature was high, but he had vomited very little. Owing to its deep position the signs were very obscure from the abdomen, but an attempt was made to reach the pus through the peritoneum. This was only in part successful, as the suppuration was already inside the psoas sheath.

Subsequently the psoas muscle was cut down upon below Poupart's ligament, and sinus forceps were passed up inside the sheath. A considerable abscess was thus opened, and the boy ultimately did well.

The close relation of an appendix abscess in this situation to the kidney, ureter, and cellular tissue of the loin, is well illustrated by this patient.

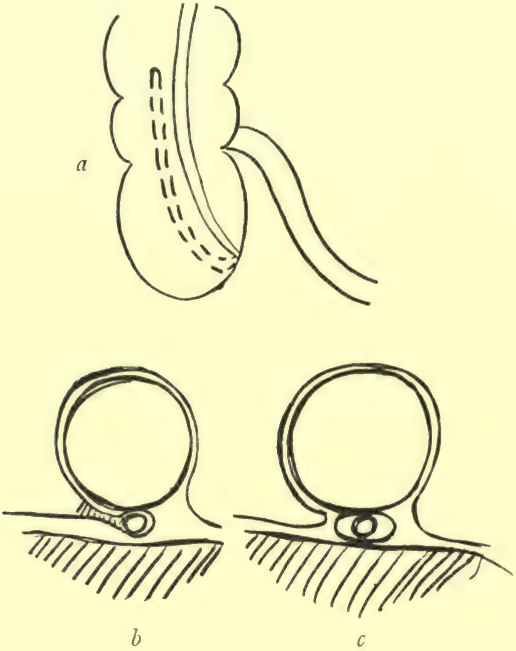


Fig. 49.—Illustrates the Manner in which the Appendix becomes Retrocæcal, and (*a*) the Position of the Retrocæcal Appendix. (*b*) The First Method is by an External Appendix lying in a Deep Sulcus between the Colon and the Loin, being shut in by Adhesions between these Two Structures; (*c*) the Second Method is by the Hernia of an Appendix into a Retrocæcal Pouch, the Mouth of which is closed by Adhesions.

He has, as you see, what appears to be a kidney incision in his right loin. When he came to my out-patients' in the first instance, he had a tender, fluctuating swelling in his right loin below the tip of the twelfth rib. He asserted that when the swelling was there he was in pain and felt ill, but that it disappeared at intervals, and he then felt better, but his urine became thick and cloudy.

The swelling was undoubtedly an abscess pointing through

Petit's triangle, and his urine, although apparently clear, showed pus corpuscles under the microscope.

He was taken into the hospital and his account confirmed. Subsequently I cut down on the abscess, and followed it through a hole in the lumbar fascia into the retroperitoneal tissues, but it then turned downward into the iliac fossa. I explored the kidney and the ureter for at least 3 inches, but could find no communication with the abscess cavity. The latter was scraped out thoroughly, except one pocket which ran down over the crest of the ilium.

The pus entirely disappeared from the urine, and did not recur. The origin of the abscess from the appendix was confirmed by the formation of a fæcal fistula some days after the operation. On one or two occasions enema fluid escaped from the wound. Before he left the hospital he had what was apparently an attack of appendicitis, the resistance and tenderness being far back in the loin. This attack cleared up rapidly when his bowels were opened. He still has a sinus, as you see, and the amount of pus discharged by this varies considerably; but as he can work well, and it does not inconvenience him, he does not wish anything further done. As the pus did not enter the pelvis of the kidney or the ureter, we must suppose it had tracked down the latter, and that the abscess opened into the bladder.

In some instances the pus of a retrocæcal appendix may form a perinephritic abscess, or even extend upwards behind the peritoneum until it reaches the non-peritoneal surface of the diaphragm, where, between the layers of the coronary ligament of the liver, it will form a subphrenic abscess which is extraperitoneal.

The treatment of the retrocæcal form of appendix abscess, as you will perceive, differs in each case. Some are best approached across the peritoneum by an incision running back towards the loin; the adhesions between the colon and the parietal peritoneum are then broken down and the abscess drained.

Some require the incision below Poupart's ligament, as already described, and others a posterior retroperitoneal incision, as in the boy I have shown you. If a subphrenic abscess forms, it is drained across the pleura in just the same manner as though it were the more common intraperitoneal form.

LECTURE III

GENERAL DIFFUSE PERITONITIS DUE TO APPENDICITIS*

THE subject of general peritonitis in appendicitis is of the greatest possible interest and importance, for practically all the cases which die from appendicitis do so because general peritonitis has supervened. If we wish, then, to diminish our mortality, we must do so by studying the manner in which general peritonitis is produced, in order that we may if possible prevent its origin. When once general peritonitis is established, the prognosis is exceedingly grave, but I shall hope to tell you of a method which gives some prospect of success even in this serious condition of affairs.

Nomenclature.—The term "general peritonitis" has been used in a very vague and indefinite sense, so that whilst so skilled an operator as Sonnenburg of Berlin reports that seventeen consecutive cases of this malady died under his treatment, other people claim almost any percentage of successes from 25 up to 75 per cent. It is, of course, clear that these surgeons are using the term "general peritonitis" in very different senses We must therefore begin by defining what we shall take "general peritonitis" to mean. In the first place, by "general" it is implied that every part of the peritoneum is inflamed; and whilst this is a condition which can only be certainly confirmed at an autopsy, yet we may diagnose it if every part of the peritoneum which is accessible to our touch is tender and painful. By the term "diffuse" we imply that the peritonitis is not localized, as it is in the case of appendix abscesses. There is no line of demarcation between the area of peritonitis around the appendix and the rest of the abdominal cavity, as there is in the latter condition. In the condition of general peritonitis no lymph barrier has been formed, and it is this barrier which circumscribes the inflammation in the more favourable termination of appendicitis. Many people hold that general peritonitis must be suppurative, but this is not at all necessary, for, as I shall point out to you later, the worst forms of peritonitis are those in which no pus is formed.

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* Delivered at the Polyclinic on March 6, 1903.

Signs and Symptoms.—We will now consider the signs and symptoms on which a diagnosis of *general diffuse peritonitis* can be made. The entire abdominal wall is tender on palpation. This tenderness extends not only over the entire anterior surface of the abdomen, but also back into the loin at the sides as far as the point at which the peritoneum is reflected on to the colon. The abdominal wall is as a rule rigid, and there is no movement on respiration. It presents, moreover, a smooth aspect, with well-marked veins, which is very characteristic of general peritonitis. Distension is present in the worst cases, and when it is due to paralysis of the gut by the inflammatory process it is, as you will easily appreciate, a most serious sign. But in many cases the distension is due to obstruction of the intestine by the matting of the coils of gut around the appendix, and when this is the case it is not of such serious import. The late Mr. Greig Smith drew attention to a most valuable sign of general peritonitis. He recommended that the abdominal wall should be explored methodically with a stethoscope for five minutes, and if during the whole of this time no gurgle was heard in the abdominal cavity indicating a peristaltic movement of the gut, then it might be safely concluded that the whole of the intestine was paralyzed. This sign I have found of the greatest value in many doubtful cases. The face in general peritonitis is of a greyish colour and sunken. It presents a great contrast to the flushed face of pneumonic diseases. In general peritonitis the eyes are sunken and surrounded by rings. The tongue is as a rule small, shrivelled, and dry. The teeth are covered with sordes. We may largely attribute the condition of the tongue and mouth to the fact that the secretion of saliva is practically suppressed in consequence of the great deficiency of fluid in the body, which at the same time makes itself evident in the shrivelled tongue and the sunken eyes. Vomiting is practically always present ; it is profuse and persistent, and towards the end it may become dark-coloured, and even fæculent in odour. The pulse is as a rule as rapid as 130 to 160 per minute. In some few cases of general peritonitis the pulse-rate is below 100 per minute, and yet at the operation the entire peritoneal cavity is found full of pus. I believe the explanation of this surprising condition of things is that the leucocytes which form the pus have so effectually stopped up the stomata of the peritoneum that practically no absorption of toxins is going on. I shall, however, refer to this process in more detail when dealing with the pathology of peritonitis. The temperature in general peritonitis is little if at all raised. Indeed, the severity of the condition may be rather taken to be inversely as the height of the temperature, so that in really serious cases the temperature is subnormal. It is only at the onset of the attack, and whilst the process is still localized, that we ever see temperatures of 103° to 104°. The patient is practically always constipated, and will react only with difficulty to purgatives or enemata. In the treatment of general peritonitis, to which I shall draw your attention later, purgatives form an important part, and I may say now that we have come to estimate the severity of a case by the quantity of calomel required to open the bowels. If the bowels are not constipated, or are easily make to work with an enema, the case is one of good prognosis.

The Causation and Pathology of General Peritonitis.

General peritonitis is due to the invasion of the peritoneum by an army of micro-organisms advancing from the inflamed appendix. The peritoneum is provided with certain measures of defence with which it opposes, and in many cases arrests, the advance of these bacteria. We shall, then, consider in detail (1) these means of defence, and shall proceed to consider in the next place (2) the causes which lead to their failure to localize the infection and convert the case into one of an appendicular abscess. Lastly, we must consider (3) the state of affairs which results from this failure to arrest the advancing infection, in order that even when general peritonitis is present we may combat these results with (4) a rational treatment.

(1) *The Measures of Defence of the Peritoneum.*—When a perforation suddenly occurs in an appendix, and a few minims of pus are thrown into the peritoneal cavity, the micro-organisms in that pus would within a very few hours spread to the farthest recesses of the peritoneum were they not opposed by the measures to which we are about to refer. Practically immediately, the neighbouring peritoneum pours out a copious flood of *antibacterial serum.* A similar effusion of serum is seen when infection in the end of the tibia or femur is threatening to invade the knee-joint, or in the pleural cavity when a subphrenic abscess is threatening

to perforate the diaphragm. In the course of some hours *the leucocytes* begin to be poured out in vast numbers from the subperitoneal vessels, and to permeate the pools of serum which are resisting the advance of the micro-organisms. These leucocytes appear to act in several ways in arresting and limiting the infection. They are probably phagocytic in action, and actually devour some of the micro-organisms. But in another way they act even more powerfully in protecting the system from the toxins of the micro-organisms by stopping up one by one the stomata in the peritoneum. A healthy peritoneum is so perforated by stomata which lead from its cavity into the neighbouring lymphatic and bloodvessels that it may be compared very aptly to a sieve, and it is capable of the most rapid absorption. If this absorption were to continue after the invasion by the micro-organisms, in a few hours the patient would die poisoned by a massive dose of toxins; and, indeed, this does occur in rare instances. But first delayed by the antibacterial serum, and then attacked by the leucocytes, the formation of toxins is retarded, and their absorption is finally prevented by the blocking up of all the neighbouring stomata by the leucocytes, so that the wall of the cavity in which the pus is contained becomes practically impermeable. The last means of defence of the peritoneum is the formation of *the lymph barrier ;* behind the two lines of defence to which we have already referred, the intestines are matted together by masses of living lymph, which consolidates them into a barrier well adapted to resist the advance of the infection. For the formation of this barrier two or three days are necessary, but after that time it is rapidly consolidated into definite adhesions, and in the course of a week or so the lymph is actually vascularized, and a layer of living tissue circumscribing the infection is absolutely complete. On the side of the barrier towards the appendix the peritoneum has been given over to the micro-organism, and the serum and leucocytes in that area are now converted into a pond of pus swarming with bacteria. But all egress from this circumscribed space is practically blocked, and we have indeed a localized abscess. The general peritoneal cavity behind the barrier is now safe from infection, and if serum has been poured out there, it is now re-absorbed without passing on to the formation of pus.

In cases of general peritonitis all these means of defence have broken down, and the infection has advanced across the peritoneal cavity. We accordingly find *different varieties of general peritonitis* *according to the degree of the failure in the defence.* In some cases the lymph barrier has actually been formed, and the process localized, when some indiscreet exercise or some unwise purgative has broken down the lymph barrier and allowed the virulent pus to flow through into the open peritoneum beyond. If the serum and the leucocytes are poured out, but no lymph barrier is formed, then suppurative peritonitis spreads all over the peritoneum. The formation of pus is comparatively a favourable sign in diffuse peritonitis, and cases in which it is formed are not so fatal as those in which the serum is poured out; but there is no diapedesis of leucocytes, either because the process is too rapid or because the patient fails to react. In these cases we find blood-stained serum, which is only a little turbid; not a trace of lymph is seen upon the intestines, and the surface of these is roughened and eroded. So that not only are the stomata not occluded, as they are in a suppurative case, but the very endothelial covering of the peritoneum is removed, so that the most rapid absorption of the toxins takes place from the peritoneum. In such cases of peritonitis we therefore find the signs of profound toxæmia; the pulse is usually as rapid as 170 and extremely feeble; the patient is cold and collapsed, and death supervenes within a few hours. A few cases have been recorded where even the effusion of antibacterial serum broke down, and where a dry peritonitis, running its course in twenty-four to thirty-six hours, carried the patient off with signs of the most profound toxæmia.

(2) *The reasons why the serum, the leucocytes, and the lymph barrier, fail to arrest and localize the peritonitis are as follows:*

In the first place, the dose of pus which suddenly enters the peritoneal cavity is probably the most important cause. Where the micro-organism has to grow through the wall of the appendix the invasion is of necessity slow, and there is ample time for measures of defence; but where an appendix distended with pus suddenly perforates, and a drachm or so of virulent pus enters the peritoneal cavity, there is great risk that it may not be localized. In general peritonitis cases, accordingly, we nearly always find a perforated appendix.

The character of the micro-organism is also a most potent factor. Some micro-organisms are of such extreme virulence that they can perforate the wall of a living appendix, and can then spread far and wide through the peritoneum, overcoming all resistance. Some twelve months ago I operated on such a case of appendicitis, where the appendix did not contain a calculus nor was there any stricture; but the whole structure, though still alive, was acutely inflamed, and the process had extended throughout the whole peritoneal cavity. In cases such as this we must regard the cause of the general peritonitis to be the virulence of the micro-organism. In other cases the continuous spread of the micro-organism appears to be due to its power to dissolve the lymph laid down as a defensive measure by the peritoneum. Thus in another case I had under my care some time ago, on which I operated forty-eight hours after the onset of the attack, I found the peritonitis fairly restricted to the right iliac fossa, but there was no formation of lymph. The appendix was removed, and the area of peritonitis packed off with gauze; but although the man lived eight days the peritonitis did not cease to spread steadily during the whole of that time, and at the post-mortem we found a total absence of lymph in the peritoneal cavity, and even in that part of it which I had packed off with gauze.

The position of the appendix in relation to the mesentery of the small intestine appears to me to be an important cause of general peritonitis. When the appendix lies behind the mesentery and the small intestine, any abscess it may form is readily localized against the posterior abdominal wall. But when it lies in front of the mesentery and the small intestine, with its end in the central region of the abdomen, then should a perforation occur it is far from the abdominal wall, and it at once infects the whole of the central region of the abdomen, which seems to be the most dangerous and to produce the greatest degree of shock. In only two cases have I definitely localized the appendix in front of the mesentery and the small intestine, and in both these instances general peritonitis resulted when the appendix became inflamed.

In some cases the fault appears to lie with the patient. There are some people who appear to react very inefficiently to bacterial invasion, and these people are often some of the strongest and most athletic members of society. One has often noticed in hospital life how the big athlete appears to be susceptible to every form of bacterial invasion. He suffers from hospital sore throats, from whitlows, and catches all the infectious diseases which he encounters in the hospital; whilst some anæmic and apparently feeble student will go through his entire course at the hospital without a single day off duty for ill-health. In the same way patients exhibit very different powers of reaction to peritoneal infections. Closely connected with this subject is the interesting question of the immunity which the peritoneum acquires after one attack has been safely passed. It is the absence of this immunity which makes a first attack so much more fatal than any subsequent one, and this fatality, as we have seen, is due to the greater frequency of general peritonitis in first attacks. The immunity of the peritoneum after the first attack may be partly explained by the formation of adhesions which mechanically localize the process from the very start; but there is no doubt that the immunity is in part at least actual, and that the peritoneum is more or less indifferent to the presence of the micro-organism. It is possible that in these cases the serum which is poured out is of higher antibacterial and antitoxic action. A common cause of the failure of the lymph barrier to circumscribe the peritonitis is the occurrence of trauma, and this is of great practical importance, as it shows the necessity of keeping all cases of appendicitis absolutely quiet. Everybody must have known of cases of appendicitis which progressed favourably for two or three days, in which the patient then got out of bed, relapsed, and died of peritonitis shortly after. In these cases we can only suppose that the movements broke down the lymph barrier and allowed a flood of pus to pour through the rent into the general peritoneal cavity. In many cases a similar result has followed the administration of a violent purge, and here, no doubt, the violent peristaltic contraction of the intestines separated one coil from another, and in this way broke the continuity of the lymph barrier. Purgatives, then, should be absolutely avoided in all localized cases of appendicitis, although, as you will see, they form a most important part of the treatment when the peritonitis has spread to all parts of the cavity.

We must now consider—(3) *The condition of things which is brought about by the failure of the peritoneum to arrest the spread of infection.* In the first place, the serum, which has been converted into pus by its invasion by leucocytes, stands about in ponds in the peritoneal pouches to which I drew your attention in my last lecture. This pus is of a most virulent character, and is charged with the toxins manufactured by the micro-organism. So virulent is it, indeed, that when in releasing it we allow it to flow over the raw margins of our wound, it kills the tissues so that they are reduced to a sloughing condition. It is an astonishing fact that a patient may hold within his abdominal cavity some pints of such a virulent fluid, and that yet his condition may not be profoundly serious.

The explanation is, as I have said, that the stomata of the peritoneum in such cases are all effectually blocked by the leucocytes, and, as we shall see later on under the head of Treatment, in emptying these collections of pus we must disturb these natural defences as little as possible. In spite of these means to prevent the virulent poisons manufactured by the micro-organism from reaching the general circulation, there is, nevertheless, in general peritonitis a very serious degree of toxæmia. This toxæmia shows itself in the ashy colour, the condition of collapse, the feeble heart, and the extreme nervous exhaustion. Another very serious result of general peritonitis is the paralysis of the coils of gut which are exposed to its action. This, fortunately, does not always occur, for it will be readily understood that when the inflammation is sufficiently severe to penetrate the wall of the intestine and paralyze it, there is little chance that the patient will recover. The paralysis of the intestine leads to a stagnation of its contents, which decompose and distend the intestine by a copious formation of gas which cannot be got rid of by the paralyzed gut. This intestinal decomposition adds to the general toxæmia. Finally—and perhaps this is the most immediate danger of general peritonitis—there is a tremendous shortage of fluid in the body. It must be remembered that, for three or four days, not only has the patient not taken any fluid by the mouth, but during that time he has expended his stock of fluid in supplying the excretion of urine, sweat, and the copious vomiting. It is well known that patients suffering from general peritonitis will lose a stone or more in weight in a few days, and this loss of weight is chiefly due to deficiency of water. We see this fact plainly indicated in the shrivelled tongue, the depressed eyes, and the drying up of the secretion of saliva in the mouth. The deficiency of fluid in the body must also have a most profound effect upon the circulation, just as it has in a similar condition in cholera.

(4) *The Rational Treatment to combat the above Results.*—First we must drain the peritoneal pouches, which are filled with ponds of pus. After having tried practically every means of doing this except evisceration, I have come to the conclusion that the rational thing to do is to empty these abscess cavities with the greatest gentleness, and with the least disturbance of the natural defences which shut their contents off from the vascular system. We went very thoroughly into the anatomy of these peritoneal pouches in our last lecture, and I then indicated that by three

incisions they might all be emptied with the least possible disturbance of their walls. I must refer you for these incisions to the previous lecture, but I may say that they are a median one above the pubes, through which the pelvic pouch and the iliac fossæ can readily be reached, and two lateral ones in either loin which give access to the subphrenic spaces on each side, and on the right also allows the subhepatic space to be efficiently drained. The appendix, if it is encountered, should be removed. I am strongly opposed to irrigating the general peritoneal cavity in these cases, for in the first place the toxin which was concentrated in the peritoneal cavity is well diluted and flushed into the general system. Then I believe the mechanical washing opens up the stomata which have been closed by the formation of lymph by the leucocytes. It often happens, moreover, that the fluid employed is not of the correct temperature, and coils of gut acutely inflamed are most sensitive to lavage with water that is too hot or too cold. As a result of some considerable personal experience of irrigation, I may say I gave it up because so many patients collapsed entirely after its employment.

Secondly, the use of boric acid. I should like to begin by saying that I have only employed this measure in two cases, but that in both these cases it was successful, and at any rate it does no harm. It seemed to me that in those cases of peritonitis in which the healthy peritoneum was not shut off from the micro-organism by lymph in a similar way the micro-organism was exposed to attacks by the surgeon, could he discover an antiseptic which would not also prove fatal to the patient. Boric acid naturally presented itself as fulfilling these conditions. Even in water it does not form more than a 5 per cent. saturated solution, and it is certainly far less irritating to living tissues than the virulent toxins produced by the micro-organisms upon which it acts. I accordingly had long sausage-shaped bags made of gauze, the end of which, for a couple of inches or so, was packed with boric acid powder; the whole was sterilized by heat. In two cases, one of which was diffuse peritonitis without adhesions, I planted these bags of boric acid in the various pouches of the peritoneum, the tail of the bag acting as a gauze drain. After three or four days, when the bag is pulled out, it is found to be empty, and I believe that in both these cases, although suppuration was not stopped, the abscess cavity more rapidly cleaned up, and they healed more quickly. A more extended experience is, however, necessary before speaking definitely upon this subject. At any rate, as I have said, it does no harm, and it is well known that small quantities of boric acid in solution will inhibit the growth of micro-organisms even when it is not strong enough to kill them.

The third means of treatment we are employing at the London Hospital for general peritonitis cases is to administer a grain of calomel every hour until the bowels are opened, and then to maintain a moderate diarrhœa. In some cases as much as 40 and 50 grains of calomel have been administered, and in one case 72 grains were given. Mr. Lawson Tait years ago advocated the use of saline purges in general peritonitis, but he gave the preference to magnesium sulphate. I prefer calomel, not only because it is easily administered and the patient cannot vomit it, but also because I believe that, given in small and repeated doses, it is the most powerful antiseptic of the entire alimentary canal that we possess. For instance, it is well known that calomel administered in small doses repeatedly to children produces grass-green motions. This result appears to be due to the action that calomel has exerted on the butyric acid fermentation in the alimentary canal. The biliverdin of the bile therefore passes from the liver to the rectum without being reduced to bilirubin, as is usually the case. The reason for administering purges is to prevent the dilatation and paralysis of the gut, with the consequent decomposition of its contents, to which I have already referred.

Finally, we employ massive saline subcutaneous transfusions, and it is the introduction of this line of treatment at the London Hospital which has entirely changed our point of view with regard to cases of general suppurative peritonitis. Previously we regarded these cases as practically hopeless, and approached them with a feeling of helplessness which was very depressing. It was only because now and then a case recovered, in spite of all expectations, that we continued to offer the parents this forlorn hope. I may say that we now approach cases of general peritonitis with a considerable feeling of hopefulness, and with a sense that a good fight will probably save our patients. I must tell you first how this treatment was brought to my notice. The chief assistant to Professor Kocher of Berne was visiting the London Hospital, and I was asked to show him round the wards. As we passed from one part of the hospital to another, we compared notes on the surgical matters that had interested us lately, and I asked him if in Berne they knew any cure for general suppurative peritonitis. He was enthusiastic about massive saline transfusion, which he told me he had adopted successfully in two cases. He described to me his apparatus, and told me that he had transfused twenty or thirty pints by four needles placed in the subcutaneous tissues. The apparatus employed by him is a somewhat complicated one, but I show you here to-day a simple and practical form, which is also inexpensive, and which can be obtained and used successfully by anyone. About 4 feet of rubber tubing, weighted at the end, carries the saline from an ordinary ewer to a glass T-tube, and this is connected by two further lengths of rubber tubing with two stout brandy-syringe needles which can be readily passed below the skin of the thighs. The saline should be of the strength of a teaspoonful to the pint (0·6 per cent.); the water should have been boiled, and in the ewer should remain at a temperature of about 115° F. The ewer should be scalded out before use, and the whole of the tubing should be boiled. To establish a siphon action, it is only necessary to put the whole of the tubing into the water in the ewer, and then, having clipped the end, to draw out the T-tube and needles over the end. The ewer should be placed at the height of 1 foot above the thighs of the patient. If a greater height than this is employed, the distension gives rise to considerable pain. With this elevation 2 pints of saline will flow into the subcutaneous tissues in an hour through the two needles. It is, of course, well to clean up the thighs and to insert the needles with all antiseptic precautions, but as long as ordinary care is employed it is not well to waste time in such serious cases in elaborate precautions. I must admit that one or two cases have developed abscesses at the point of puncture, but these cases were of such a serious character that they may consider themselves fortunate to have lived long enough to develop these abscesses, and the latter were, after all, of a very mild subacute character, and healed up without the least trouble. The quantity of saline infused in this fashion must vary considerably with the size of the patient. A full-grown man can safely take 15 pints in the first twenty-four hours after the operation. In two little boys of seven or eight whom I recently treated in this way, we infused 4 pints directly after the operation, and 4 pints a few hours later. Eight pints is a moderate quantity. The transfusion is repeated day by day as occasion demands, unless the vomiting ceases and the patient can drink freely, or the rectum will absorb a pint six times a day. The effect of this transfusion is really very wonderful; the whole face of the patient fills out, the rings round the eyes diminish, or may even disappear, the tongue becomes larger and moist as the secretion of saliva is once more established, the pulse is greatly improved and becomes slower and stronger. In some cases I have seen infusions increase the flow of pus from the abdominal tubes. This saline method is based on many sound reasons. In the first place, as has been pointed out, the body is very short of fluid, and it is a well-known fact in biology that all protoplasm, in order to perform its functions well, must be distended with water. I show you here two flowers, one of which I removed from water some hours ago. You will see that it is drooping and faded, but if I replace it in water, within an hour or two it will look as fresh as its companion. In the same way, when a human being is deprived of water he fades and droops, and, as I have pointed out to you, our peritonitis cases are not only taking no fluid for three or four days, but they are constantly losing water from their system through many channels. They cannot drink to replace this water, and saline infusions into the rectum do not always act well, more especially as we wish to keep up a persistent diarrhœa by means of calomel. We can then replace the water that they have lost, and maintain a continued supply of it, by these two needles placed beneath the skin of their thighs. In the second place, these massive transfusions alter the relation between the vascular system and the pus in the peritoneum. Whilst the vascular system is depleted, there is every inducement for fluid to pass from the ponds of pus in the peritoneum into the vessels around. But when by means of ten or fifteen pints of saline we have thoroughly filled up the vascular system, not only is there less inducement for the toxins to be absorbed into the blood, but a flow tends to be established in the opposite direction; and, as I have told you, some cases in which abscesses in the peritoneal cavity were being drained by tubes have had an increased flow from those tubes when saline transfusion was established.

In the third place, the transfusion dilutes the toxins circulating in the blood, and probably in this way diminishes the virulence of their action on nervous tissues, on heart muscle, and on the other tissues of the body. In the last place, this flood of saline passing through the body carries out with it, especially through the kidneys, these toxins in a very dilute form, and with the least possible damage to the renal epithelium.

In speaking of the results of this system of treatment, it is very difficult to speak definitely, for the difficulty which we encountered at the beginning of this lecture, in defining what should be called "general peritonitis," makes the collection of a series of cases which would stand that test extremely difficult. And when one is enthusiastic about any form of treatment, there is a very natural tendency to sweep into the results less severe cases. I will therefore simply state that in five successive cases of peritonitis under my care, in which the inflammation was practically general, only one died; and in this case death occurred one hour after the operation, and before the method of treatment could be got into running order. A colleague of mine who is employing the same line of treatment has had only one death in six cases of general peritonitis. One of his cases, I think, is deserving of especial note. The boy was admitted to the hospital not only distended, but with fæcal vomiting. He required 72 grains of calomel in hourly doses to open his bowels and to keep them open. He was transfused 26 pints in all—on, I believe, four occasions of about 7 pints each.

I can definitely state that this method of simple drainage, combined with calomel and massive transfusions, has in my experience proved the most hopeful measure I have yet encountered in the treatment of general diffuse peritonitis. This method has, moreover, made it possible to prolong cases of peritonitis to a point at which two recovered by an actual crisis. This phenomenon, I believe, has not previously been observed, chiefly because, hitherto, patients have died of toxæmia and deprivation of fluid, and were not able to fight to a finish the actual battle with the micro-organism. In one of my cases, a little boy of eight years of age, we removed the appendix and drained the pelvic abscess, but found the peritoneum generally infected. By means of the treatment I have indicated we kept him alive for ten days, during the whole of which time he was vomiting, his temperature was slightly raised, and the edges of the wounds remained dry and glazed. At the end of these ten days, one afternoon he had a most curious attack of delirium. His pulse fell from 132 to 72 per minute in twenty-four hours; the vomiting ceased, he began asking for solid food, and the wounds in the abdomen took on a healthy appearance.

In a similar case of mine, in a girl aged seventeen, the pulse fell from 124 to 96 in twenty-four hours, her temperature came down, and she rapidly convalesced. The occurrence of these crises impresses on us the lesson that ultimately the battle will be fought out between the man and the micro-organisms in the inaccessible recesses of the peritoneal cavity, and that in some cases the micro-organism may gain the upper hand. No treatment will, therefore, always be successful in general diffuse peritonitis, but whilst the battle is still undecided, and whilst the result is still in the balance, there is no reason why our patient should die of depletion of fluids or of paralytic dilatation of his intestine.

SURGICAL ASPECTS OF SUBPHRENIC ABSCESS *

Definition.—For the purposes of this paper any localized collection of pus which is in contact with the under surface of the diaphragm is called a "subphrenic abscess."

It is not always customary to include hepatic abscesses under the heading of "subphrenic," but inasmuch as they frequently, thin out the hepatic substance so that their entire upper boundary is formed by the diaphragm, and they then develop along similar lines and require like treatment, there appears to be no good reason why they should not be classed as subphrenic abscesses, as they certainly are (Fig. 50).

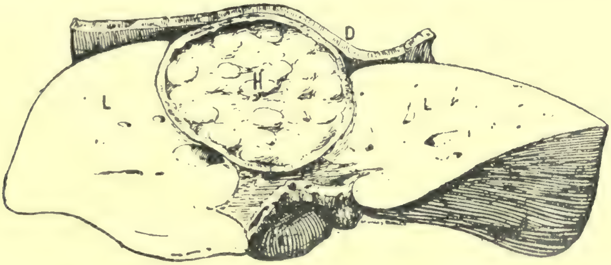

FIG. 50.—FROM A SPECIMEN IN THE LONDON HOSPITAL PATHOLOGICAL DEPARTMENT, SHOWING HOW HYDATIDS AND ABSCESSES OF THE LIVER BECOME "SUBPHRENIC."

H, Hydatid; L, liver; D, diaphragm.

The Anatomical Subdivisions of Subphrenic Abscesses, with their Special Etiology, Signs and Symptoms, and Treatment.—The chief object of this paper is to emphasize the importance of the anatomical subdivisions of subphrenic abscess.

The under surface of the diaphragm is marked out into four peritoneal spaces and two cellular ones (Fig. 51). The four peritoneal spaces are separated from one another by the cruciform arrangement of the ligaments of the liver—namely, the coronary, falciform, and the right and left lateral ligaments. The falciform ligament divides the subphrenic space into two parts, right and left. Each of these is again subdivided into a larger anterior and a smaller posterior part by the corresponding lateral ligament. The four peritoneal subphrenic fossæ may therefore be classified and named as follows :

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A. Intraperitoneal—
- I. Right :
  - 1. Anterior.
  - 2. Posterior.
- II. Left :
  - 3. Anterior.
  - 4. Posterior.

The two extraperitoneal cellular spaces are also right and left, so that the classification may be completed—

B. Extraperitoneal—
- 5. Right.
- 6. Left.

A right extraperitoneal subphrenic abscess is between the layers of the coronary ligament, which it separates and opens out, whilst the left originates in the perinephric tissues around the upper end of the left kidney, and afterwards strips the peritoneum off the under surface of the left dome of the diaphragm.

These spaces and their corresponding abscesses will now be defined in more detail.

1. *A Right Anterior Intraperitoneal Subphrenic Abscess.*

Anatomy.—This abscess is situated between the upper surface of the right lobe of the liver below, and the dome of the diaphragm above. The falciform ligament limits it towards the middle line, whilst behind it passes back to the right lateral ligament of the liver. In front it is shut off from the peritoneal cavity by a line of adhesions which varies somewhat in position. When the appendix infects the cavity from the outside and behind, the anterior margin of the liver becomes adherent to the diaphragm in front, and forms the lower boundary of an abscess which occupies the upper and posterior part of the fossa, and incidentally it may be pointed out that these adhesions prevent the liver from descending as the abscess grows.

When, however, the fossa is infected from below and in front by a perforating gastric or duodenal ulcer, the line of adhesions is as a rule formed by the great omentum and transverse colon, and the whole of the fossa is filled with gas and pus, whilst the liver is separated from the diaphragm and pushed upwards and backwards.

This fossa has very important connections at its posterior and outer part, where it communicates round the margin of the liver and round the extremity of the right lateral ligament with the subhepatic pouch which we are about to describe, whilst at the same point it is in continuity with the right lumbar fossa, which lies between the cæcum and ascending colon and the loin.

These connections are of great importance, for not only does infection usually reach both these fossæ from the appendix (Fig. 52) along the right lumbar pouch, but also pus which has originated in the fossa from the liver, stomach, or duodenum, may afterwards spread to its two connections and pass down the loin into the pelvis. This association should be recognized and anticipated, and prevented by a loin incision when it threatens. I have notes of such a case in which the right anterior subphrenic abscess was due to a perforating pyloric ulcer. It was drained from the right hypochondrium, but subsequently spread insidiously to the subhepatic pouch and down the loin to the pelvis, and led to the death of the patient three weeks after the operation.

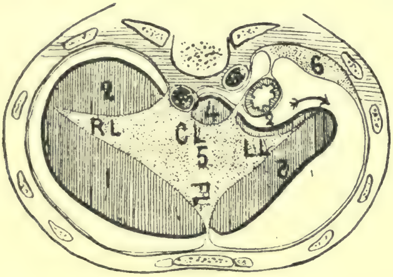

FIG. 51.—DIAGRAMMATIC VIEW OF THE UPPER SURFACE OF THE LIVER EXPOSED BY REMOVING THE DIAPHRAGM.

CL, Coronary ligament; FL, falciform ligament; RL, right lateral ligament; LL, left lateral ligament; 1, right anterior intraperitoneal space; 2, right posterior intraperitoneal space or subhepatic fossa (note it crosses the middle line to the left under liver margin); 3, left anterior intraperitoneal space; 4, left posterior intraperitoneal space or small sac of peritoneum; 5, right extraperitoneal space; 6, left extraperitoneal space.

Incidence and Associations.—Right anterior subphrenic abscess occurred 27 times in 76 cases at the London Hospital. In 11 cases the abscess was simple, and in 15 cases one other fossa was involved: in 9 cases the right posterior intraperitoneal fossa, in 4 the right extraperitoneal subphrenic space, and in 2 the left anterior fossa. In 1 case two other fossæ contained pus—namely, the small sac of the peritoneum and the right extraperitoneal cellular space. It is therefore clear that in every case where a right anterior subphrenic abscess has been opened these other fossæ, but especially the subhepatic fossa, should be examined daily, lest pus should insidiously collect in them.

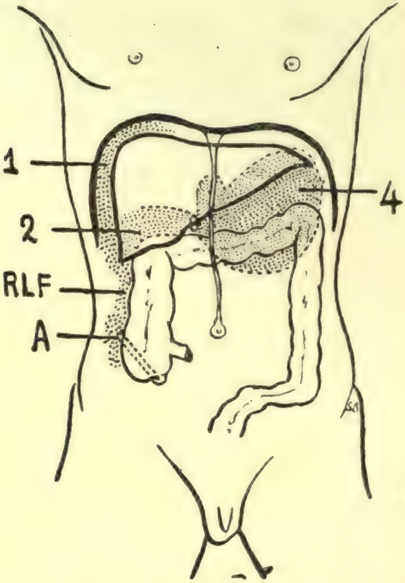

FIG. 52.—THE SPREAD OF SUPPURATION FROM AN EXTERNAL APPENDIX, A, UP THE RIGHT LUMBAR FOSSA, RLF, TO THE LIVER MARGIN, WHERE IT INFECTS 1, THE RIGHT ANTERIOR INTRAPERITONEAL SPACE, AND 2, THE SUBHEPATIC FOSSA.

From the latter suppuration is shown invading 4, the small sac of the peritoneum through the foramen of Winslow, a rare event.

Etiology.—The causes of the 27 abscesses in this group were as follows:

10 Appendicitis; one of these had pylephlebitis (*q.v.*).
7 Liver abscesses: 3 suppurating hyatid; 1 tropical abscess; 1 pylephlebitis due to appendicitis; 1 suppurative cholangitis due to gall-stones; 1 cause not found.
4 Perforated gastric ulcers.

2 Anterior perforating duodenal ulcers.
1 Ruptured intestine.
1 Pyosalpinx.
1 Pyæmia.
1 Gall-stones; this case had suppurative cholangitis (*q.v.*).
1 Followed pneumonia.
1 In no cause was found.

In nearly all cases due to appendicitis the appendix was external in position and occupied the right lumbar fossa, but in one case the tip of the appendix reached to the subhepatic fossa, and in another lay between the liver and the diaphragm.

Other causes are stated to be ruptured liver, pancreatitis, and cancer of the pylorus and colon.

Special Signs and Symptoms of Abscess in the Right Anterior Subphrenic Space.—Clinically this abscess occurs in three forms:

1. Simple infection from behind.
2. Infection from in front.
3. Infection combined with the subhepatic pouch.

In the first of these forms (Fig. 53), in which the cause is nearly always appendicitis, the liver is firmly adherent to the diaphragm in front, and the pus is localized at the upper and back part of the recess. As the abscess grows it expands the diaphragm above, and gives rise to well-marked signs of compression and inflammation at the right base. The signs in the right hypochondrium and epigastrium are very slight, if any, because the liver cannot descend. Local signs will be present as usual in proportion to the size of the abscess.

In the second class of cases (Fig. 54) a gastric or duodenal ulcer has usually perforated, and free gas has escaped into the peritoneum. The liver becomes separated from the diaphragm and drops upwards and backwards. The abscess in typical cases presents, in the hypochondrium and epigastrium, as a triangular area, bounded by—(1) The costal margin. (2) A line joining the ensiform cartilage to the umbilicus. This margin is due to the falciform ligament, and is convex to the left, because the ligament is pushed over by the abscess. (3) Below, a band of rigidity and tenderness stretches from the umbilicus to the lower costal margin. It is from 1 inch to 2 inches wide, and is placed over the limiting adhesions and peritonitis. The area of the triangle in a fully-formed case bulges forwards, fluctuates, and is not very tender. Above, in the epigastric angle, signs of a gas bubble can usually be found where liver dulness should be present. It is circular in outline, hyperresonant, amphoric sounds are heard over it, and it passes round the side to the back as the patient is rolled over.

In the third class of cases (Fig. 53), which are usually also appendicular, both intraperitoneal fossæ on the right side are simultaneously infected. The liver is adherent to the diaphragm, as in the first class, but the subhepatic abscess bulges forward from below its margin, and forms an abdominal swelling, occupying the right hypochondrium and running back into the loin as low as the iliac crest. In short, the signs of a subhepatic abscess are simply superadded to those described under Class 1.

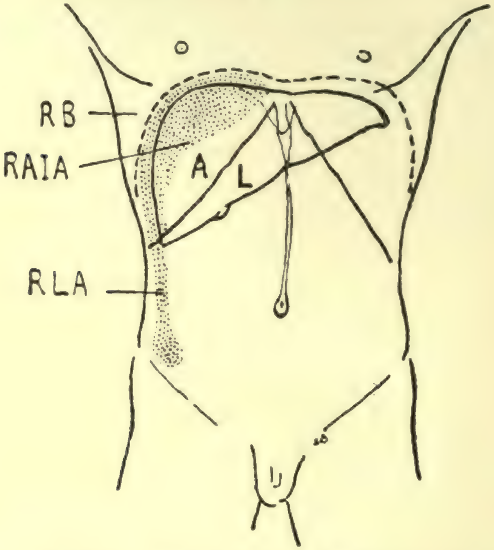

Fig. 53.—The Abdominal Signs in the Posterior or Appendicular Variety of Right Anterior Intraperitoneal Subphrenic Abscesses.

The liver, L, does not project nor move on respiration, because its lower and anterior part, A, is adherent to the diaphragm. If a right lumbar abscess, RLA, is present, there will be tenderness and rigidity in the flank. The pus is localized in upper and back part of the fossa, RAIA, and gives signs at the right base, RB.

The Treatment of Right Anterior Intraperitoneal Subphrenic Abscesses.—In appendicitis, when the infection is recent and the abscess is diffuse, the right loin incision should be employed. In recent cases due to perforated gastric and duodenal ulcers which present in the epigastrium, an anterior incision should first be made, and when the perforation has been closed a counter-opening may be made in the loin. When the abscess has become well localized, after ten or fourteen days, it is better to drain it by the posterior transpleural or subpleural method, whether it presents in the epigastrium or not, if the exploring needle can find pus in the lower intercostal spaces behind. The liver should be pressed up by an assistant, so as to close the pleural space, whilst the pleura is incised. Where the exploring needle fails to find pus behind, and an epigastric swelling is present, the anterior epigastric incision should be employed, and the peritoneum opened as high up as possible in the subcostal angle.

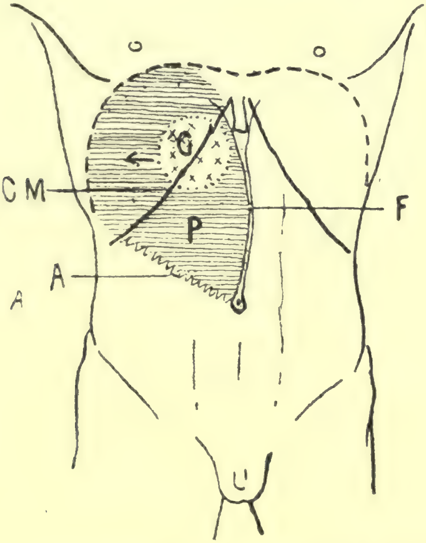

FIG. 54.—THE ABDOMINAL SIGNS IN THE ANTERIOR OR GASTRIC OR DUODENAL VARIETY OF RIGHT ANTERIOR INTRAPERITONEAL SUBPHRENIC ABSCESS.

The abscess produces the "diagnostic triangle" in the right epigastrium. F, Falciform ligament bulging to the left; A, band of adhesions from umbilicus to costal margin, broad, tender, and rigid; CM, costal margin; P, pus in contact with abdominal wall, bulging, fluctuating, dull, scarcely tender; G, gas bubble over liver dulness, causing tympanitic, amphoric, and coin sounds—moves round chest as patient is rolled over.

2. *A Right Posterior Intraperitoneal Subphrenic Absces*

Anatomy.—The fossa in which this abscess forms is better known as the "subhepatic pouch" or the "right renal fossa." It is a pyramidal space transversely disposed beneath the overhanging margin of the liver. Its base and most capacious part rests against the right lateral abdominal wall, projecting just below the last rib. The apex is formed by the upward slope of the margin of the left lobe of the liver. The liver and gall-bladder are its anterior boundaries throughout. The posterior wall is divided into a right and left part by the projection forward of the lumbar vertebræ in the middle line. The right part of the fossa is capacious, and will hold about a pint of fluid before it escapes below. Its posterior wall is formed by the upper part of the right kidney, and by the lower part and crus of the diaphragm, and towards the left by the common bile-duct and duodenum. It is here that the foramen of Winslow leads into the left posterior intraperitoneal fossa (*vide* Fig. 52). To the left of the middle line the fossa is a narrow cleft between the left lobe of the liver in front, and the small omentum and upper and anterior surface of the stomach behind. Above the subhepatic fossa is bounded to the right by the right lateral ligament of the liver, and to the left

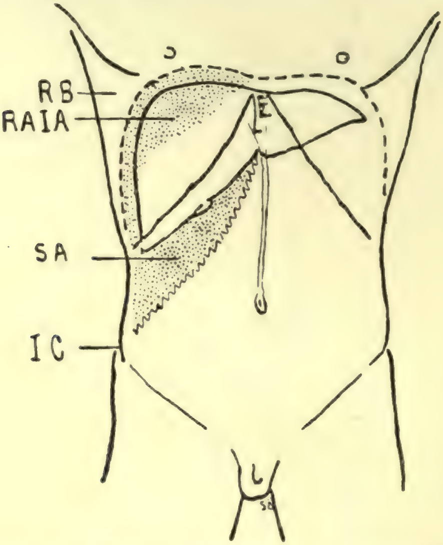

FIG. 55.—THE ABDOMINAL SIGNS WHEN A RIGHT ANTERIOR INTRAPERITONEAL SUBPHRENIC ABSCESS IS COMBINED WITH A SUBHEPATIC ABSCESS, AS OFTEN OCCURS IN APPENDICULAR CASES.

The former, RAIA, produces signs of compression and inflammation at the right base, RB. The latter, SA, produces a triangular swelling below the liver margin, limited towards the abdomen by a band of tender rigid adhesions running from the ensiform, E, back into the right loin, usually as low as the iliac crest, IC. Pus or gas are sometimes found in contact with the abdominal wall within the triangle.

by the transverse fissure. Below, the boundary is formed on the left by adhesions between the margin of the liver and the anterior surface of the stomach, and on the right by similar adhesions of the great omentum and transverse colon to the margin of the liver and the anterior abdominal wall. This fossa is continuous at its base with the right lumbar pouch and with the right anterior subphrenic space. At the middle of its posterior wall the foramen of Winslow connects it with the small sac of the peritoneum, whilst its apex is but feebly shut off from the left anterior subphrenic space (*vide* Fig. 51).

Incidence and Associations.—In 76 cases this fossa contained pus in 10, and always in association with some other pouch—in 9 cases with the right anterior subphrenic space, and in 1 case with the left anterior subphrenic space.

Etiology.—The causes of suppuration in this fossa in the 10 cases which occurred out of 76 subphrenic abscesses at the London Hospital were as follows:

6 Appendicitis.
1 Gastric ulcer.
1 Duodenal ulcer.
1 Pneumonia.
1 Bronchiectasis.

Other causes are liver abscess and suppuration in connection with the gall-bladder and bile-ducts, especially after operative procedures.

The signs that the subhepatic fossa is involved in suppuration are diffuse tenderness and rigidity spreading from below the right costal margin, and running downwards and backwards into the right loin, usually as low as the iliac crest (*vide* Fig. 55). As this abscess has so great a tendency to be combined with others, the signs of a right lumbar abscess, or of a right anterior intraperitoneal subphrenic abscess, must be looked for in addition.

Treatment.—This abscess is best drained by a loin incision as far back and high up as possible below the last rib. The index-finger guides the drainage-tube transversely below the liver. Should the signs at the right base indicate the presence of pus under the dome of the diaphragm, the index-finger should be inserted between the liver and the diaphragm, and a long tube passed up into the right anterior intraperitoneal subphrenic space.

3. *A Left Anterior Intraperitoneal Subphrenic Abscess.*

Anatomy.—This is also known as an "anterior perigastric abscess" or a "perisplenic abscess" (Fig. 56). This fossa is bounded by the diaphragm above. The left lobe of the liver is below and to the right. The spleen is to the left, whilst adhesions of the anterior surface of the stomach and the great omentum with the abdominal wall limit it below. To the right it is sharply bounded by the falciform ligament. Behind, it runs back as far as the left lateral ligament of the liver. This fossa is continuous to the right with the subhepatic pouch below the left margin of the liver, and below with the left lumbar pouch, which lies between the descending colon and the loin. Another fossa, which I believe has not been previously described, connects it with the pelvic fossa. It lies deeply in the left lumbar recess (Fig. 57). To the right it is bounded by the vertebral column. The descending colon bounds it to the left, whilst it is roofed in by the back of the mesentery. Below, over the left sacro-iliac joint, it communicates with the recto-vesical or recto-uterine pouch. Above, it runs up to the duodeno-jejunal flexure, and there the pus passes forward in the splenic flexure to reach the left anterior subphrenic space. A narrow chain of pus can always be found in this situation in cases of general peritonitis when the mesentery is turned upwards and to the right, and this fossa is the line of infection when an ordinary pelvic appendix abscess is followed by an insidious subphrenic

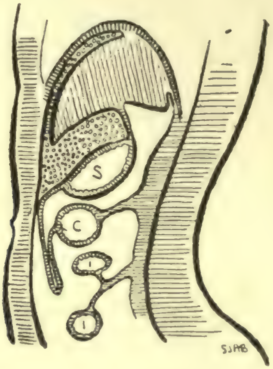

Fig. 56.—An Anterior Perigastric Abscess occupying the Left Anterior Intraperitoneal Space and the Apex of the Subhepatic Fossa, through which Pus might readily spread across the Middle Line to the Right.

abscess on the left side, as in the case of Fitzpatrick, illustrated by Fig. 58.

Case.—This man was admitted under my care at the London Hospital in January, 1903, on the seventh day of the disease, suffering from a localized pelvic appendix abscess. Pus was found in an enema the same evening, but, as the symptoms did not diminish, the abscess was opened by a suprapubic incision next day, and the abscess was drained by a large tube passed down into the pelvic pouch.

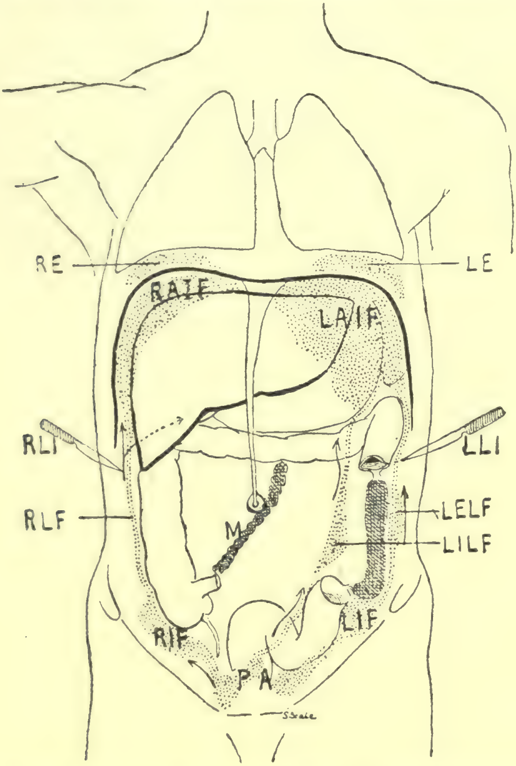

Fig. 57.—The Spread of Pus from a Pelvic Appendicular Abscess, PA, up Both Loins to the Subphrenic Spaces.

The right side has already been described under Fig. 3. Up the left side the pus may either pass by the left iliac fossa, LIF, and the left external lumbar fossa, LELF, or it may follow the left internal lumbar fossa, LILF, and reach the subphrenic space at the splenic flexure. RE and LE represent empyemata; RLI, right loin incision; LLI, left loin incision; M, mesenteric attachment.

His progress during fourteen days was not satisfactory. His tongue was dry and cracked, he had profuse diarrhœa, he wasted rapidly, and his temperature was persistently raised to 100°-101° F. No signs could, however, be discovered at the right base or in the lower part of the abdomen. After fourteen days a large swelling was found in his left hypochondriac region, exhibiting all the signs shown in Fig. 60. This swelling was incised high up in the left epigastrium, and much stinking gas and pus was evacuated from the left anterior intraperitoneal space. He appeared better after this operation, but two days later he suddenly coughed up, with profound collapse and fright, a large quantity of most foul pus and flatus, apparently from a left empyema, but no amount of needling could find the pus in the large area of dulness at the left base. After he had evacuated similar material on several occasions, he convalesced and entirely recovered.

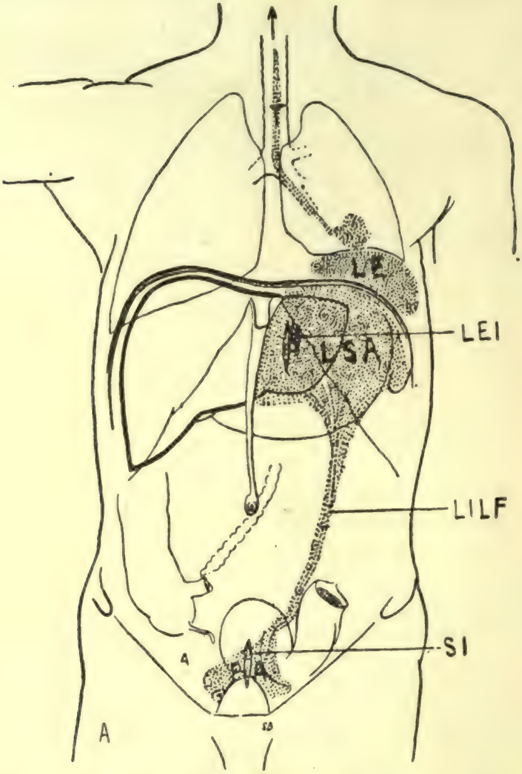

FIG. 58.—ILLUSTRATES THE CASE OF FITZPATRICK, IN WHOM PUS TRAVELLED FROM THE RECTUM TO THE MOUTH, WITH ULTIMATE RECOVERY.

A, Appendix; PA, pelvic abscess; SI, suprapubic incision; LILF, left internal lumbar fossa; LSA, left subphrenic abscess; LE, left empyema; T, trachea discharging pus and flatus; LEI, left epigastric incision.

I believe this infection spread by the left internal lumbar fossa, for on nearly every day the loins had been examined for a spread of infection, and none could be discovered external to the descending colon.

Incidence and Associations.—The left anterior intraperitoneal subphrenic fossa was involved in 30 of 76 cases of subphrenic abscess at the London Hospital, and of these 26 were simple. In 3 cases one other fossa was involved—in 2 cases the right anterior intraperitoneal, and in 1 case the small sac of the peritoneum. In 1 case two of the other pouches contained pus, namely, the subhepatic pouch and the right extraperitoneal cellular space.

Etiology.—The causes of suppuration in the 30 cases of left anterior subphrenic abscess were as follows:

16 Gastric ulcer.
3 Pyæmia and splenic infarct.
2 Cancer of stomach.
2 Resections of 1 stomach; 1 transverse colon.
1 Anterior perforating duodenal ulcer.
1 Appendicitis via pelvic abscess and left internal lumbar fossa.
1 Typhoid.
1 Parturition.
1 Hydatid of liver.
1 Cancer of pancreas.
1 Bronchiectasis via a liver abscess.

Special Signs and Symptoms of a Left Anterior Intraperitoneal Subphrenic Abscess.—The previous history is very frequently one of dyspepsia, pain after food, vomiting with relief, and perhaps hæmatemesis. The onset is usually due to a subacute or chronic perforation of a gastric ulcer. The signs of compression and inflammation of the base of the left lung and pleura are usually present. They are sometimes bilateral. The local signs over the abscess call for no special comment, except that gas is nearly always present.

An abdominal swelling is nearly always present in this variety, and in fully developed cases is very characteristic (Fig. 60). It occupies a triangular area in the left epigastrium and hypochondrium. Above, it is limited by the costal margin on the outer side. Its inner margin stretches from the ensiform cartilage to the umbilicus, and is more or less convex to the right according to the size of the abscess. This is, of course, the bulging falciform ligament. The lower margin stretches from the umbilicus to the

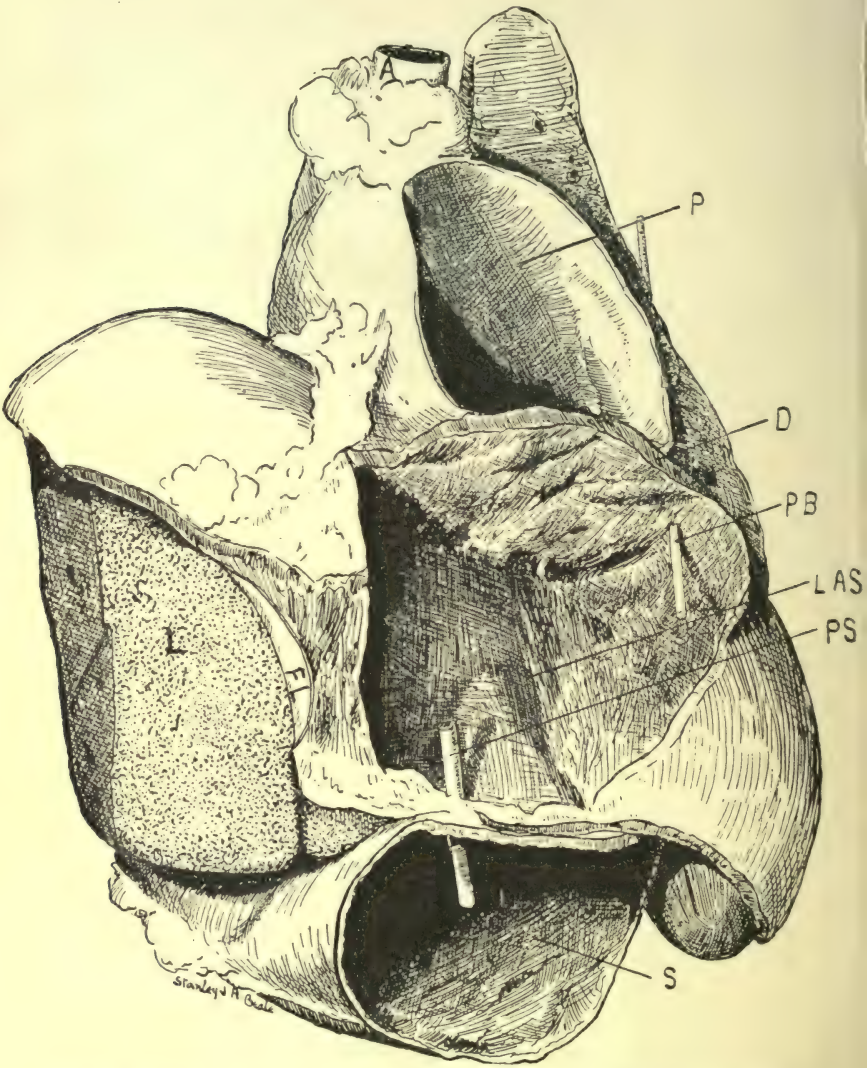

Fig. 59.—From a Specimen in the London Hospital Museum, prepared by Dr. Cecil Wall.

A left anterior subphrenic abscess, LAS, communicating with the stomach, S, through a perforation, PS. The cavity of the abscess is laid open by an incision turning up a flap of the diaphragm, D. It is limited to the right by the falciform ligament, FL. Another perforation, PB, connects the abscess through the diaphragm with a bronchus. P, Pericardium; L, liver; A, aorta.

lower part of the costal margin. It is a broad, tender, and rigid band, and corresponds to the limiting peritoneal adhesions. The area of the triangle bulges forward, may fluctuate and be dull on percussion, and is not very tender. At the upper angle, where the gas collects as the patient lies on his back, a tympanitic rounded area can be made out where liver dulness should be. Where much gas is present the whole of the triangle is tympanitic, and amphoric and coin sounds can be obtained. When the patient is rolled over, the swelling becomes dull on percussion, and the signs of a collection of gas pass under the left ribs, which were previously dull. Where the quantity of gas is not excessive, the lower part of the triangle is dull even in the supine position.

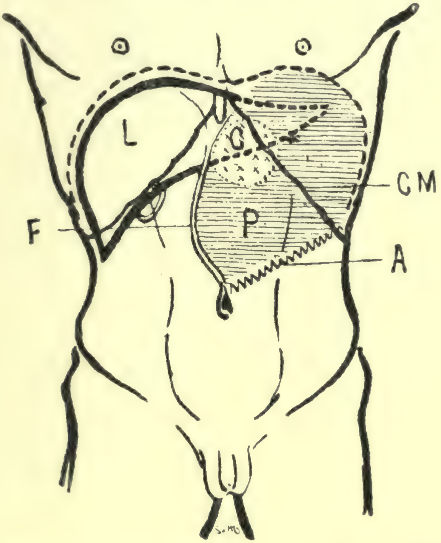

FIG. 60.—THE DIAGNOSTIC TRIANGLE OF A LEFT ANTERIOR INTRAPERITONEAL SUBPHRENIC ABSCESS.

F, Falciform ligament convex to the right; A, broad band of adhesions, tender and rigid, stretching from umbilicus to costal margin; CM, costal margin; P, bulging lower part of area of triangle, dull, and not very tender, occupied by pus; G, circular area over liver dulness, tympanitic, amphoric, and coin-sounds travelling to the left as the patient is rolled over (this is due to gas in the abscess, and is seldom absent in this situation); L, liver.

In those cases in which the abscess has burst into the stomach and freely communicates with it through a large opening, the signs are much less obvious. The abscess is never distended. The temperature is therefore very irregular, and often reaches normal. The abscess often contains at one time gas and at other times fluid, so that the signs vary frequently. In some of these cases no abdominal swelling is present, but as a rule a rigid, tender tumour, which does not move on respiration, projects from below the left ribs. Sometimes the abscess lies between the liver and the stomach, and the former is found, as usual, in the epigastric angle. These cases are prone to spread to the right beneath the liver margin, and may then track down the right loin. In splenic cases the abscess at first occupies the deep posterior recess of the fossa around the spleen, and the anterior part of the space is obliterated by adhesions ; but in nearly all cases the pus at last reaches the epigastric angle and produces a typical swelling.

Treatment.—In the acute and diffuse stage, soon after perforation, it is usual to open the abdomen in the middle line and close the perforation. Practice then differs. The abdomen is sometimes sponged out and closed altogether. In other instances a drainage-tube is passed into the pelvic fossa through a puncture above the pubes, and the patient is sat up in Fowler's position. Others drain from the anterior incision. I prefer to make a counter-opening in the left loin and pass a tube up to the spleen, and then adopt the Fowler position and the pelvic drain. When the abscess has become localized and points in the epigastrium, it should be opened by a left epigastric incision as high up as possible in the costal angle. A large tube, irrigation, suction, and position, are then usually enough to insure efficient drainage. Otherwise the abscess may be counter-opened by the posterior transpleural method, guided by a finger in the cavity.

When the abscess has not extended forward to the front, it is nevertheless well to explore it from the epigastrium, and then open it by the posterior transpleural method above the adhesions, avoiding the spleen. The lateral subpleural method is suitable to some of these cases, or a posterior loin incision may be made, and a finger passed up through the adhesions to the spleen, after the general peritoneal cavity has been packed off.

4. *A Left Posterior Intraperitoneal Subphrenic Abscess.*

Anatomy.—This sac is better known as the "small sac of the peritoneum" (Fig. 61). At the left extremity of this cavity one surface of the spleen abuts upon it. Even anatomically this pouch extends no lower than the transverse colon, and when peritonitis is present adhesions encroach still farther on the space below, and drive the pus into the upper and back part of the cavity, where it lies behind the upper and back part of the stomach and small omentum and the liver. Above, it is prolonged upwards into a narrow pouch, which lies between the posterior surface of the Spigelian lobe and the crus of the diaphragm. The upper part of the posterior wall covers the pancreas, and the upper part of the left kidney, and suprarenal.

To the right this cavity communicates through the foramen of Winslow (Fig. 52) with the subhepatic pouch, but this communication is so narrow that, as a rule, it is sealed at the first outbreak of inflammation, and as a consequence infection seldom enters the smaller sac from the subhepatic fossa, nor does it spread in a contrary direction.

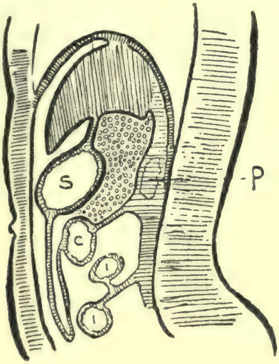

FIG. 61.—A SUBPHRENIC ABSCESS OCCUPYING THE SMALL SAC OF THE PERITONEUM.

The lower part of the cavity is obliterated by adhesions. Above, the cavity extends up in a narrow pouch between the Spigelian lobe and the crus of the diaphragm. P, Pancreas in posterior wall; S, stomach in front with lesser omentum; C, transverse colon below with its mesocolon.

Incidence and Associations.—Among the 76 cases of subphrenic abscess, the lesser sac was involved in only three instances. In one an anterior perigastric abscess was also present, and in another a right extraperitoneal, and in addition a right anterior intraperitoneal abscess occurred as well.

Etiology.—Of the 3 cases of suppuration in the lesser sac of the peritoneum, 2 were due to *posterior perforating gastric ulcers.* One case was due to a cholecystitis, due to gall-stones, which after many years' duration produced *suppuration in the glands of the small omentum.* From the loculi of pus the small sac of the peritoneum was infected. It bulged forward between the liver and the stomach, and was opened in the epigastrium and marsupialized; but at the same time the bile-duct became infected, and suppurative cholangitis led to the formation of an abscess between the layers of the coronary ligament, and another in the right anterior intraperitoneal subphrenic space.

Injuries and Inflammation of the Pancreas.—It is said that *appendicular suppuration* may reach the lesser sac through the foramen of Winslow from the subhepatic pouch. *Tuberculous peritonitis* may become localized in this cavity (Osler). It may be infected from the *liver*, especially if the *Spigelian lobe* is diseased. Finally, infarcts and abscesses in the *spleen* may spread to the lesser sac of the peritoneum.

The Signs and Symptoms of an Abscess in the Small Sac of the Peritoneum.—When the small sac of the peritoneum is the seat of inflammation and suppuration, its cavity is more or less obliterated by adhesions. The abscess may therefore completely distend the sac, as was found in one of my cases; or, on the other hand, a small collection of pus may be shut in between the Spigelian lobe and the spine, in the extreme upper part of the pouch, a condition which occurred in another of my cases. When the whole sac is filled with pus, the signs are similar to those of pseudo-pancreatic cysts. The swelling may project forward between (1) the stomach and liver, (2) between the stomach and transverse colon, (3) between the stomach and spleen, or (4) below the transverse colon. When the cavity of the sac is almost entirely obliterated by adhesions, and more especially when the abscess communicates freely with the stomach, the diagnosis is more difficult. Signs may or may not be present at the left base of the lung. The exploring needle may give definite results, but it is hard to hit a small collapsed cavity in so deep a situation. A careful consideration of the history and of the general signs of suppuration, combined with the exclusion of the presence of pus elsewhere, may justify an exploratory incision.

Treatment.—When the abscess points in front, it should be cut on and marsupialized and drained, a method which was followed in one of my cases. Posterior perigastric abscesses should be explored from the front, and drained from behind by a loin incision or the posterior transpleural route. The pouch between the Spigelian lobe and the spine must be nearly inaccessible.

5. *A Right Extraperitoneal Subphrenic Abscess.*

Anatomy.—This abscess forms in the cellular space between the layers of the coronary and other peritoneal ligaments of the liver (*vide* Fig. 51). The largest part of this space is on the upper and back part of the liver. It is continued forward between the layers of the falciform ligament to the epigastrium, where it is connected with the deep lymphatics of the abdominal wall which accompany the superior epigastric vessels through the diaphragm to the anterior mediastinal glands. I have seen an abscess between the layers of the falciform ligament which originated apparently by this route from an appendicular sinus burrowing in the abdominal wall. To the left the cellular tissue of the coronary ligament is continuous with that contained in the left lateral ligament as far as the œsophagus. To the right there is a similar extension between the layers of the right lateral ligament. Behind, the subphrenic cellular space is continuous with the retroperitoneal cellular tissue, and by this route infection reaches it from the right kidney, posterior surface of the duodenum, and retrocolic appendix cases. Hepatic abscesses very frequently extend up into this cellular space, thinning out the liver substance over them, until they touch the diaphragm and occupy the whole of the space (*vide* Fig. 50). When an abscess forms and grows in the right extraperitoneal subphrenic space, it expands its dimensions enormously, pushing the peritoneal reflexions on the liver farther and farther back, and the liver itself lower and lower in the abdomen. This is very different from the right anterior intraperitoneal abscess, in which the liver becomes adherent to the diaphragm early in the process, and cannot afterwards be pushed down, so that the abscess must expand the dome of the diaphragm upwards into the chest.

A right extraperitoneal subphrenic abscess may extend forward between the layers of the falciform ligament until it points in the middle line of the epigastrium, where it may be incised without opening the peritoneum (Fig. 62). In other cases the pus may present below the last rib; more usually, however, it perforates the diaphragm above. It is stated that this form of abscess may extend directly under the external arcuate ligament to the posterior mediastinum. Lymphatic extension may involve either pleura, and it is said that both pleuræ are more often involved than in the intraperitoneal form of subphrenic abscess. The pericardium, curiously enough, is seldom involved.

Incidence and Associations.—Of 76 cases of subphrenic abscess at the London Hospital, 19 were in the right extraperitoneal cellular space. In 4 cases another abscess was present in the right anterior intraperitoneal space. In 2 cases two other abscesses were present. In one the small sac of the peritoneum was involved, and the right anterior peritoneal space; in the other the subhepatic pouch and the left interior intraperitoneal space.

Etiology.—The causes of infection of the cellular tissue between the liver and the diaphragm may be divided into 4 groups. The 19 cases in this series of 76 were arranged as follows, and other possible causes are added in their appropriate places:

I. Liver:
  - 7 Suppurating hydatids.
  - 6 Tropical abscesses.
  - 1 Liver abscess of unknown origin.
  - 1 Cholangitis from suppurating hepatic glands from gall-stones.
  - Pylephlebitis, and pyæmia.
  - Actinomycosis.
  - Ruptured liver.

II. Right retroperitoneal cellular tissue:
  - 1 Congenital cystic kidney.
  - Other renal abscesses and perforations of the ureter.
  - 1 Posterior perforating duodenal ulcer.
  - Retrocolic appendicitis.
  - Acute periostitis of lumbar vertebræ.
  - Perforation of the common bile-duct in its lower part.
  - Suppuration in the head of the pancreas.
  - Wounds and lymphatic infections.
  - Colonic ulcers and cancer.

III. Thoracic:
  - 1 Bronchiectasis.
  - Abscess of lung.
  - Empyema.
  - Actinomycosis.

IV. Lymphatics of the anterior abdominal wall:
  - 1 due to appendix sinus.

The Signs and Symptoms of Right Extraperitoneal Subphrenic Abscess.—The previous history will be that of the cause, which in many cases is some disease of the liver. The signs of the abscess are not preceded by those of perforative peritonitis, as is usually the case in intraperitoneal forms. The onset is therefore more often insidious and indefinite, and jaundice is often an early symptom. Signs of compression and inflammation are found at the base of the right lung, and sometimes also on the left side. The liver is pushed down from below the costal margin, often as low as the umbilicus, and it moves on respiration (*vide* Fig. 62). The tumour so formed may be tender, but there is not the same rigidity and signs of peritonitis which are found in intraperitoneal forms. This abscess may pass forward between the layers of the falciform ligament and present in the epigastrium as a fluctuating swelling. In rare instances it may reach the tissues of the right loin and present there. This abscess seldom contains gas.

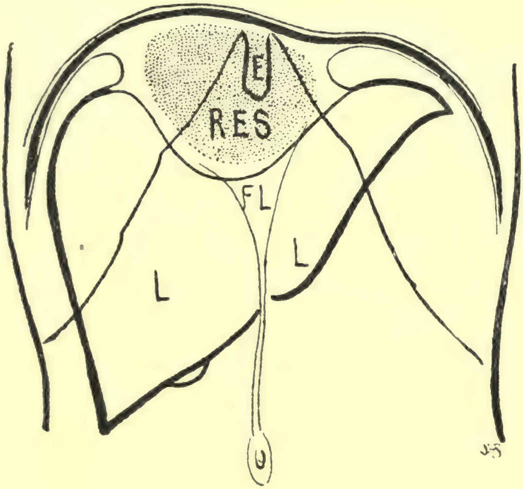

Fig. 62.—The Signs of a Right Extraperitoneal Subphrenic Abscess, RES, pointing in the Epigastrium below the Ensiform Process, E, and having separated the Layers of the Falciform Ligament, FL, so that the Abscess may be opened extraperitoneally.

Note that the liver is well pushed down in this case, and freely movable. Compare Fig. 53.

Treatment.—When the abscess points in the epigastrium, it should be opened there by an incision strictly in the middle line. In two of my cases I was able to reach the abscess without opening the peritoneum. With a large drainage-tube, irrigation, suction, and position, counter-drainage is seldom necessary. If the abscess does not point in front, it will probably be diagnosed by puncture between the ribs behind, over the area of dulness. The rule is then to follow the needle and adopt the posterior transpleural method. Should the pus point in the right loin, the abscess should be opened there, and a tube be passed up into the subphrenic space.

6. *The Left Extraperitoneal Subphrenic Abscess.*

Anatomy.—The retroperitoneal cellular tissue is as abundant in the left loin as it is on the right side. It contains the left kidney and suprarenal body, the pancreas, descending colon, and the left lumbar lymphatic glands. There is, however, no large cellular space below the diaphragm upon the left side equivalent to that upon the right. For this reason, when suppuration does occur in the left retroperitoneal cellular tissue, it shows a much stronger tendency to appear in the loin as a left lumbar abscess. In a few cases, however, such an abscess strips the peritoneum from the under surface of the diaphragm on the left side, and forms a large collection there.

Incidence and Associations.—This has occurred four times in 76 cases of subphrenic abscess at the London Hospital, and in none of these cases was any other subphrenic abscess present.

Etiology.—The cause in two of these cases was acute periostitis of the transverse process of the first or second lumbar vertebra. The third case originated in a localized empyema at the left base. The fourth was probably due to a posterior perforating gastric ulcer, and Mr. Mayo Robson has described a similar case.

Other causes are similar to those narrated on the right side, under the headings of Retroperitoneal Cellular Infections and Thoracic Infections.

The Signs and Symptoms of Left Extraperitoneal Subphrenic Abscess.—The signs are those of an abscess pointing in the left lumbar region, associated with compression and inflammation at the left base of the lung. In cases which have been overlooked the pus may reach the left iliac fossa.

Treatment.—A left loin incision over the swelling will efficiently drain the pus away.

Pathology.—The under surface of the diaphragm is subdivided into four peritoneal fossæ and two cellular spaces. Infection of one or more of these can be produced by the passage of micro-organisms from the viscera which lie in them, or by extension to them from fossæ which are in continuity with them.

Abscesses which occupy the four peritoneal fossæ are examples of localized peritonitis. Pathologically they originate as acute infections of the peritoneum. A profuse but diffused exudation of serum is poured into the fossæ, accompanied by the symptoms of acute peritonitis. After a day or two the serum becomes turbid with leucocytes. The stomata in the peritoneum become blocked by proliferation of endothelial cells, and by leucocytes forming a layer of lymph over them and limiting absorption. The margins of the area of infection begin to be defined about the fourth day by a living lymph barrier, which localizes and contains the forming abscess. At first the purulent contents are diffused in the irregular spaces and cracks between the organs, but as the lymph barrier converges and contracts upon it, gluing the organs firmly together, the pus is driven into one more or less rounded, well-defined collection. This change is not complete until about the end of fourteen days. This change from a diffuse exudation about the fourth day to a localized abscess from the eighth to the fourteenth day has important practical applications. The transpleural method of drainage, in which a hole is made through the diaphragm into the abscess from above, is most unsuitable for the treatment of the early and diffuse stage of subphrenic suppuration. An abdominal incision (usually in the loin) should be employed in such cases, and a long tube should be passed under the diaphragm from below. At a later stage, when the abscess is well shut off under the diaphragm, the abdominal route is less suitable, for in the first place the liver may be torn in separating it from the diaphragm (*vide* Fig. 65), or some hollow viscus may be opened; or, on the other hand, pus may be admitted to the general peritoneal cavity.

When the collection of purulent fluid is first localized about the eighth or ninth day beneath the dome of the diaphragm, it may be small in quantity, and deeply placed, far from the surface, and covered by important organs and cavities. The nature of all abscesses, however, is to increase in size. The diaphragm is driven upwards and outwards so that the lower part of the pleural cavity is obliterated, and often closed by adhesions, and the lung is pushed up. The viscera which bound the abscess cavity below are pressed downwards are far as their adhesions will permit, so that the abscess can now be opened at a lower and lower spot on the thoracic wall, should the transpleural operation be adopted, or it may even appear as a fluctuating swelling in the epigastrium directly beneath the abdominal wall. For these reasons it is often advisable to await the growth of a well-localized subphrenic abscess, from, say, the eighth to the fourteenth day. This evolution of a subphrenic abscess is best illustrated by pus in the right anterior intraperitoneal space (*vide* Fig. 65). When this follows an appendicitis, it is often at first a small localized collection about the size of a tennis-ball, beneath the dome of the diaphragm on the right side, and opposite the sixth or seventh intercostal spaces in the line of the angle of the scapula, and covered by the lung. It cannot well be opened from below, for the liver would probably be torn in separating it from the diaphragm. If the transpleural route be adopted, the pleura must almost of necessity be opened about the seventh space, and the lung collapsed, and even then it may not be found possible to drag the diaphragm up to the chest wall, and fix it with stitches before opening the abscess. This actually happened in one of my early cases, in which I tried to evacuate a diffuse subphrenic collection by a transpleural route, after resecting the seventh rib in the scapular line on the fourth day. It is therefore better to wait three or four days until the abscess has spread down to the level of the ninth or tenth space, and has obliterated the pleural cavity and pushed up the lung.

When the infection is in one of the two cellular spaces, the pathological processes which produce the abscess are those of lymphangitis and cellulitis. The lymph spaces of the cellular tissue become distended, first with serum, and then with leucocytes. The centre of the area of infection is liquefied, necrosed, and converted into pus. In the meantime the reaction of the tissues around has produced the granulation tissue barrier which demarcates and contains the collection of pus.

The Reason why Pus tends to collect beneath the Diaphragm.—It is usually stated that the causes are the suction action of the thorax, especially during inspiration, and the normal circulation of lymph from the peritoneum through the stomata of the diaphragm to the pleural cavities. The recent investigations of surgeons in connection with the treatment of diffuse peritonitis have shown that the main cause of subphrenic accumulations is gravitation. As a patient lies flat upon his back in bed, the posterior extremity of his subphrenic spaces are only separated from the mattress by the skin, the ribs, and the diaphragm, which together do not make up much more than ½ inch of tissues. The pouch of Douglas, or the recto-vesical pouch in the male, in a similar way, are only separated from the mattress by the skin, the coccyx, and the width of the flattened rectum. Between these two dependent pouches the thick muscles and tissues of the loins, with the kidney and the perinephric fat, rise as two elevated mounds. When the general peritoneum is infected, a profuse flow of serum is poured out, and this gravitates upwards into the subphrenic spaces, and downwards into the pelvic pouch. If the patient is well propped up on pillows, or is supported by an efficient bed-rest (Fowler's position, *vide* Fig. 63), this fluid will run from beneath the diaphragm over the loin mound into the pelvic pouch, from which it may be drawn off by a tube passed to the bottom of that fossa from a puncture above the pubes. This method is only effective in preventing subphrenic abscesses whilst the exudate is diffused. When it is shut in and localized, special measures must be adopted to evacuate it.

The Micro-Organisms found in the Pus of Twelve Subphrenic Abscesses.—The examination of the pus was carried out in the Pathological Department of the London Hospital by Dr. William Bulloch :

Sterile	3 cases.
Bacillus coli communis	3 ,,
Staphylococcus aureus	2 ,,
Streptococcus	1 case.
Pneumococcus	1 ,,
Bacillus pyocyaneus	1 ,,
Bacillus typhosus	1 ,,

Many other micro-organisms have been recorded by other writers.

The Bursting of Subphrenic Abscesses.—Twenty-three of 76 cases ruptured spontaneously. They were distributed as follows:

Into a bronchus	4 cases.
Into the pleura (right)	2 ,,
Into the pleura (left)	3 ,,
Into the general peritoneum	1 case.
Into the stomach	8 cases.
Into the intestine	1 case.
Into the colon	2 cases.
Through the skin—one at the umbilicus, and another in the right hypochondrium	2 ,,

Some of these cases demonstrate the possibility of a natural cure, but experience shows how extremely rare this is.

The Age of Seventy-five Cases of Subphrenic Abscess.—I have arranged 75 cases of subphrenic abscess in which the age was stated in decades, and the result is as follows:

0–10	10–20	20–30	30–40	40–50	50–60	60–70
6	5	29	15	13	6	1

The curve is no doubt due to the age incidence of the chief causes—namely, gastric ulcer, appendicitis, and hepatic suppurations, which, as already stated, account for two-thirds of the cases.

The Sex of Seventy-six Cases of Subphrenic Abscess.—Of the series of 76 cases of subphrenic abscess at the London Hospital, 1900-1907, 43 were males (56·6 per cent.), and 33 were females (43·4 per cent.), so that sex does not appear to exercise any great influence.

The General Etiology of Subphrenic Abscesses.—The following organs occupy the fossæ which abut upon the under surface of the diaphragm: The stomach and duodenum, liver and bile-passages, pancreas and spleen, and the upper end of the kidneys. In the event of perforation or suppuration occurring in these organs, the infection will spread directly to the peritoneal or cellular space in which the organ lies.

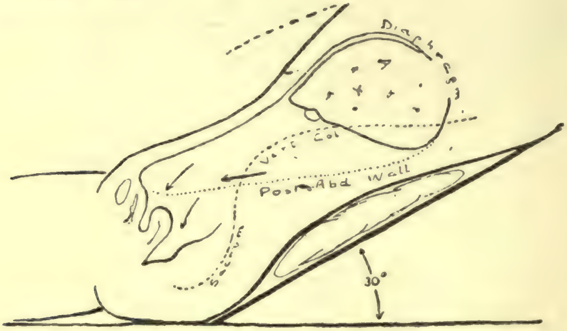

FIG. 63.—"FOWLER'S POSITION," TO PREVENT PURULENT SERUM GRAVITATING INTO THE SUBPHRENIC SPACES IN CASES OF DIFFUSE PERITONITIS. (From Kelly's "Appendicitis.")

The arrows indicate how in the sitting-up posture fluid flows into the pelvic pouch, from which it can be drawn off by a tube passed through a suprapubic puncture.

On the other hand, the infection may originate in the lower part of the abdominal cavity, such as in the small intestine, the appendix, or the pelvic viscera. The infective process then spreads along those peritoneal or cellular fossæ which at their upper end communicate with the spaces which abut upon the diaphragm (*vide* Fig. 57). I have analyzed the causes of 76 consecutive cases of subphrenic abscess which have occurred at the London Hospital in eight years, and have arranged them in order of their frequency:

Gastric ulcer	21 cases.
Appendicitis	12 ,,
Suppurating hydatid	8 ,,
Tropical abscess of liver	6 ,,

Duodenal ulcer	5 cases.
Parturition	3 ,,
Pyæmia	3 ,,
Splenic infarct	3 ,,
Thoracic	3 ,,
Cancer of stomach	2 ,,
Acute periostitis of transverse process of lumbar vertebræ	2 ,,
Operations, resections	2 ,,
Suppurative cholangitis	1 case.
Pylephlebitis	1 ,,
Cancer of pancreas	1 ,,
Typhoid	1 ,,
Pyosalpinx	1 ,,
Ruptured gut	1 ,,
Gall-stones, cholangitis	1 ,,
Congenital cystic kidney	1 ,,
No cause found	2 cases.

NOTE.—There is a little overlapping in this table.

It will be seen that perforating gastric and duodenal ulcers are responsible for about one-third of all cases. Appendicitis is responsible for one-sixth, and hydatid and tropical abscesses of the liver for another sixth between them, whilst the remaining third of the cases is distributed among many causes, each of which is individually rare.

The Routes of Infection and Localization of Subphrenic Abscesses due to Gastric Ulcer.—Twenty-one cases of subphrenic abscess out of 76 were due to gastric ulcer, and these contain examples of the localization of pus in all the six subphrenic spaces but one.

1. In 16 cases the perforation was on the anterior wall of the stomach, and near the lesser curve. The septic matter escaped direct into *the left anterior intraperitoneal space*, and the abscess was localized there.

2. In 1 case the margin of the left lobe of the liver formed such firm adhesions to the anterior surface of the stomach that the infection was directed to the right into *the subhepatic pouch.*

3. *The right anterior intraperitoneal space* was involved in 4 cases. One of these was a further extension from the subhepatic pouch of the case described under (2). In the other 3 cases perforation occurred near the pylorus, and the septic matter and gas passed direct into this fossa. The liver became separated from the diaphragm, and dropped upwards and backwards, so that the whole of the space was filled with gas and pus, which presented in the right hypochondriac region.

4. In 2 cases a posterior perforating gastric ulcer infected the *lesser sac of the peritoneum.*

5. In 1 case the cavity of the lesser sac was obliterated by adhesions. The ulcer then invaded and infected the *left extraperitoneal cellular space*, where a large abscess formed. Mr. Mayo Robson* has recorded a similar case.

6. Finally, a perforating gastric ulcer may produce pylephlebitis, and the branching abscesses in the liver may infect the *right extraperitoneal cellular space*, or the right anterior intraperitoneal fossa.

In cancer of the stomach the infection follows a similar course, and localizes in like situations.

The Routes of Infection and Localization of Subphrenic Abscesses due to Appendicitis.—Of the 12 cases of subphrenic abscesses due to appendicitis, in the series of 76 cases, the pus was localized as follows:

Right anterior intraperitoneal fossa ...	10 cases.
Right posterior intraperitoneal fossa ...	6 „
Left anterior intraperitoneal fossa ...	1 case.
Right extraperitoneal fossa	1 „

Two fossæ were involved in 6 cases.

Appendicitis may infect the subphrenic fossæ in four ways:

1. As a part of an acute general peritonitis, but these cases are not included here because they are clinically so different from the localized abscesses which are the subject of this paper.

2. By a more or less slow and direct extension up the lumbar peritoneal fossæ from the pelvis.

3. Through the medium of the portal vein, as a part of pylephlebitis.

4. By lymphatic extension (*a*) up the right retroperitoneal cellular tissue, or (*b*) up the lymphatics around the deep epigastric artery to the falciform ligament.

In the majority of cases the appendix lies external to the cæcum and ascending colon, and reaches a variable distance up the right lumbar fossa, towards the liver margin: (1) When it becomes inflamed the infection passes into the subhepatic fossa (6 cases), and (2) in practically all these cases, and in some others in addition, the sepsis also extends between the liver and the diaphragm into the right anterior intraperitoneal space (10 cases). (3) It is stated that pus may pass from the subhepatic fossa through the foramen of Winslow to the lesser sac of the peritoneum, but no such case has occurred at the London Hospital. (4) A localized pelvic appendix abscess may escape up either loin. When it does so on the left side, it spreads up either the internal or external lumbar fossa to the left anterior intraperitoneal fossa. One such case occurred in my series, and the details of this extension are more fully described under the heading of that fossa (*vide* Figs. 57 and 58).

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* "Diseases of the Stomach," 1904, p. 320.

5. A retrocæcal or retrocolonic appendix may infect the right retroperitoneal cellular tissues, either as a diffuse cellulitis or as a lymphangitis. In either case the infection may spread to the extraperitoneal cellular space between the layers of the coronary ligament, or to the lesser omentum and its glands.

6. In one of my cases in which cellulitis of the abdominal wall followed an appendicitis, an abscess formed in the epigastrium between the layers of the falciform ligament. Apparently extension had taken place along the lymphatics around the deep and superior epigastric arteries.

7. Pylephlebitis is not uncommon in appendicitis, and when this occurs the abscesses in the liver frequently infect the right extraperitoneal space and the right anterior intraperitoneal fossa. I have seen both these spaces infected in this manner, where the appendicitis was due to a pin in the appendix.

It will be seen that all the subphrenic spaces and fossæ may be infected by appendicitis, except the left extraperitoneal cellular space.

The Routes of Infection and Localization of Subphrenic Abscesses due to Liver Disease.—Of the series of 76 cases of subphrenic abscess at the London Hospital, 18 were due to liver diseases. Under this heading are grouped suppurating hydatids, tropical abscesses, pylephlebitis, cholangitis, and liver abscesses due to some other cause.

The subphrenic abscesses which resulted were localized as follows:

Right anterior intraperitoneal	7 cases.
Right posterior intraperitoneal ...	1 case.
Left anterior intraperitoneal	1 „
Left posterior intraperitoneal	0 „
Right extraperitoneal	15 cases.
Left extraperitoneal	0 case.

As a liver abscess enlarges it tends to thin out the liver substance over it and come to the surface. In the majority of cases this occurs between the layers of the coronary ligament, which are pushed aside until the diaphragm forms a considerable part of the upper boundary of the abscess (*vide* Fig. 50).

The cellular tissue of the right extraperitoneal space may, however, be infected from quite a small abscess in the liver, especially when this is due to pylephlebitis or suppurative cholangitis, and in these cases the subphrenic abscess and the hepatic abscess are separate. A hepatic abscess may reach the surface of the liver where it is covered by peritoneum, and it may then either actually rupture or infect the corresponding peritoneal fossa by transudation of micro-organisms. In this way any of the intraperitoneal fossæ may be infected, but a reference to the above list will show that this is only common in the case of the right anterior intraperitoneal space.

The Routes of Infection and Localization of Subphrenic Abscesses due to Gall-Stones.—Only one such case is included in my series of 76 subphrenic abscesses. In this case suppurative cholecystitis led to abscesses in the glands of the small omentum, and from these the lesser sac was infected and suppurative cholangitis was produced. The suppurative cholangitis led to a right extraperitoneal subphrenic abscess, and to a right anterior intraperitoneal subphrenic abscess. Another case has recently come under my notice in which a stone impacted in the diverticulum of Vater led to distension and rupture of an ulcer on the right side of the neck of the gall-bladder. The septic bile poured into the subhepatic pouch and was shut off below, but the infection spread round the edge of the liver into the right anterior intraperitoneal fossa, which was also occupied by an abscess. It is also said that a gall-stone impacted in the lower third of the common bile-duct may infect the retroperitoneal cellular tissues by actual perforation or transudation of micro-organisms, and by this route reach the right extraperitoneal space.

The Routes of Infection and Localization of Subphrenic Abscesses due to Duodenal Ulcers.—This series of 76 cases of subphrenic abscess contains 5 due to duodenal ulcer, and these were localized as follows:

Right anterior intraperitoneal	1 case.
Right posterior intraperitoneal ...	2 cases.
Left anterior intraperitoneal	1 case.
Left posterior intraperitoneal	0 ,,
Right extraperitoneal	2 cases.
Left extraperitoneal	0 case.

The most important distinction is into anterior and posterior perforating ulcers. An anterior perforating duodenal ulcer nearly always directly infects the subhepatic pouch in which it lies, but it is easy for the infection to reach the right anterior intraperitoneal fossa round one of the margins of the liver. A duodenal ulcer is, however, as a rule so close to the pylorus that it is practically in the middle line, and consequently the pus may pass to the left of the falciform ligament and enter the left anterior subphrenic space.

When the ulcer is posterior it may infect the retroperitoneal cellular space. This occurred in 2 cases of my series.

The Route of Infection and Localization of Subphrenic Abscesses due to Thoracic Causes.—Only three such cases occurred in my series. It is a very remarkable fact that, whilst nearly all subphrenic infections pass readily through the diaphragm to the pleuræ, lungs, or pericardium, it is a very rare circumstance for the contrary to occur. The causes of this are: (1) That the intrathoracic pressure is negative, whilst the intra-abdominal is positive; (2) because the lymphatic flow through the diaphragm is always ascending; and (3) probably the lymphatics of the diaphragm are valved upwards. In 1 of the 3 cases referred to above, an acute lumbar pneumonia of the right base was followed by a pneumonic abscess in the right anterior and posterior intraperitoneal fossæ. This was presumably a lymphatic infection.

In another a localized empyema at the left base burrowed into the left extraperitoneal cellular space, passing below the external arcuate ligament. In the third case a bronchiectasis of the right base in a child perforated the diaphragm into the liver. The liver abscess so formed ruptured into the subhepatic fossa, and further infected the left anterior intraperitoneal space.

The Symptoms of Subphrenic Abscesses.—The diagnosis of a subphrenic abscess is not really a difficult matter when the physical examination is conducted in an orderly and complete manner. To achieve this it is well to divide the examination under several headings:

I. The history of (*a*) diseases which may produce a subphrenic abscess, and (*b*) the character of the onset.

II. The general signs and symptoms which indicate that suppuration is going on somewhere in the body.

III. Abdominal signs and symptoms.

IV. Thoracic signs.

V. Under the heading of local signs will be considered the direct evidence of the abscess itself.

I. *The History in Subphrenic Abscesses*—(*a*) *Of the Cause.*—Of 21 cases of subphrenic abscess due to gastric ulcer, a previous history of dyspepsia was obtained in 17. The patients complained of pain after food, relieved by vomiting, and in several instances of hæmatemesis.

In the duodenal cases the pain had been in the right hypochondrium and had occurred some two hours after meals, and some stated that the pain was relieved by taking further food, and in several melæna was noted. Of the 12 cases due to appendicitis, a history of previous attacks was obtained in only two, no doubt because appendix abscesses nearly always occur in the first attack. The cases due to tropical abscess of the liver gave a history of foreign residence without exception, and in nearly every instance stated that they had suffered from dysentery, malaria, or enteric fever. It is interesting to note, however, that three patients who had resided in the tropics suffered from subphrenic abscess from other causes than liver abscess—namely, cancer of the pancreas, acute periostitis of the left transverse process of the second lumbar vertebra, and pyæmia after a confinement. A history of jaundice was recorded in 10 of 15 cases due to hepatic abscesses of various kinds, and this symptom did not occur in any other case. One patient who developed a left subphrenic abscess in a blood-clot which formed after a resection of the transverse colon for a new growth gave a previous history of hæmophilia. Three patients dated their trouble from a septic confinement, and two from an attack of enteric fever.

The duration of the symptoms when the case came under observation was noted in 48 of 76 patients. This period varied from one day to sixteen months, and averaged eight weeks, but nearly all the cases due to perforative peritonitis were admitted ten to fourteen days after the onset.

(*b*) *The Nature of the Onset of Subphrenic Abscesses.*—Of 76 cases of subphrenic suppuration, in 40 the onset was *sudden*, and in 36 more or less *insidious*. The chief importance of this distinction is that the great majority of intraperitoneal subphrenic abscesses begin by an acute perforation into the peritoneum, whilst, on the other hand, extraperitoneal collections of pus below the diaphragm result from a spreading cellulitis, and are therefore slower in their beginnings. Twelve cases of subphrenic abscess due to appendicitis were all acute in onset. Of 23 cases due to gastric ulcers and cancer, 15 were acute in onset, and 8 were insidious. The only 2 cases in the whole series due to cancer of the stomach were insidious in their onset. The other 6 cases were due to chronic gastric ulcers which formed adhesions before they perforated the wall of the stomach. Of the 5 cases of subphrenic abscesses due to duodenal ulcer, 3 were anterior in position and acute in onset; 2 cases were posterior, and insidious at the beginning, and in both these cases melæna was present. Of 8 suppurating hydatids of the liver, 6 were slow in onset; 1 was suddenly infected, and 1 ruptured into the peritoneum, so that the onset of symptoms was instantaneous. Of 7 hepatic abscesses, 6 were subacute in onset, and in only 1 was the commencement described as "sudden." The one subphrenic abscess due to cancer of the pancreas began acutely. When a subphrenic abscess is secondary to septicæmia or pyæmia, the origin is subacute, and the same remark appears to apply to those cases which are secondary to splenic abscesses.

Pain is nearly always the first symptom of the onset of a subphrenic abscess. It was noted in 56 of 76 cases, and was referred in every case to the situation where the abscess afterwards formed. In cases due to perforative peritonitis, it was sudden, severe, and stabbing, whilst in extraperitoneal cases it was gradual in onset and aching in character. In 4 gastric cases the pain suddenly supervened after a full meal.

Vomiting is the next symptom in point of frequency and time, and occurred in 29 of 76 cases. It is almost entirely restricted to those subphrenic abscesses which originate in a perforative peritonitis, and especially to gastric and duodenal cases.

Nausea, *hiccough*, and *collapse*, were described in a few histories of the onset of subphrenic abscesses. In 8 of 76 cases it was stated that the acute onset was followed by *a period of obstinate constipation*, no doubt due to the peritonitis, but, as will be pointed out later, as the abscess becomes well localized septic diarrhœa nearly always appears.

Hæmatemesis and *melæna* bear a dual relation to subphrenic abscesses. In the majority of cases they are due to hæmorrhage from a gastric or duodenal ulcer, which subsequently perforates, and are a part of the previous history; but in a few cases they arise from a chronic and insidious subphrenic abscess which has ruptured into the stomach, and has then become acute in its progress. In the latter case they are described under the onset.

II. *The General Signs and Symptoms of Suppuration.*—In nearly all cases of subphrenic abscess an examination of the general symptoms has made it clear that a collection of pus was present in some part of the body, and when the rest of the body, including the pelvis and thorax, had been excluded, it was probable that the abscess was beneath the diaphragm. These septic symptoms were as follows: The patient's *tongue* was dirty, and often dry and cracked. His face was *pale*, *anæmic*, and muddy, an appearance which I have elsewhere described as the *purulent complexion*. The majority of cases were covered in a *profuse sweat*, which soaked the pillow, and were *weak*, *listless*, *short of breath*, *and had no appetite* for their food. Some suffered from a great *thirst*. They were *thin* and *emaciated*, and had lost from 1 to 2 stone in weight since the onset of their illness. This *wasting* is a very constant sign. The majority suffered from a profuse septic *diarrhœa*, but not a few continued *constipated*.

Pyrexia was always present. As a rule it was low (100° to 101°), sustained, and rocked night and morning. In several cases, however, it frequently reached normal, and was never above 100° F. These cases were well localized, or had ruptured into the stomach or intestine. On the other hand, in the diffuse and acute stages temperatures as high as 104° to 105° F. were often recorded.

The pulse-rate per minute varied according to the degree of localization of the abscess. In many cases it was no faster than 70 to 80, and as a rule was about 100, but in acute cases it often reached 130 or 140 per minute.

Rigors occurred in 10 of 76 cases, and of these 6 died, so that this is a very fatal symptom. In 5 pyæmia was present, but 2 of these were very chronic cases, and recovered. In 2 a perigastric abscess freely communicated with the stomach. One was due to suppurative cholangitis, and 1 to acute periostitis of the transverse process of the second lumbar vertebra.

Parotitis was present in 1 gastric case, which did not live.

Petechiæ and other cutaneous signs of pyæmia were present in 2 cases, of which 1 succumbed, but the other recovered.

Albuminuria was present in 1 case, and *delirium* and *coma* in another.

Leucocytosis was noted in all the cases examined. In 2 cases it was as low as 11,000 per cubic millimetre. In 2 cases a differential count was made, and in one of these the polymorphonuclear cells numbered 71·5 per cent., and in the other 87 per cent.; in the others it is simply stated they were in great excess.

Leucocytosis appears to be a most valuable sign, and it is to be regretted that it was not sought for more frequently in this series.

Eosinophilia was noted in 2 of 3 cases of subphrenic abscess due to hydatids of the liver in which it was looked for.

III. *The Abdominal Signs and Symptoms of Subphrenic Abscesses.—An abdominal swelling* was described in no less than 50 of 76 cases of subphrenic abscesses. In 33 of these the swelling was due to the abscess and the peritoneal adhesions round it. In the remaining 17 cases the swelling was the liver, but in 14 of these a hepatic abscess was present. In the remaining 3 cases, in one the edge of the liver only was felt; in another a Reidel's lobe was present; and in the third case the pus was in the right extra-peritoneal cellular space. When the pus was located between the liver and diaphragm within the peritoneum, the liver was not pushed down in a single instance, because it was adherent to the diaphragm along its lower margin, as has already been explained under Pathology.

The abdominal swelling formed by a subphrenic abscess does not descend on respiration, because it is adherent. The part of the swelling formed by peritoneal adhesions is rigid, tender, and dull on percussion. The part formed by pus in contact with the abdominal wall is scarcely tender, bulges and fluctuates, and is dull on percussion. Where gas is present in the abscess, a tympanitic area is formed, as a rule, high up in the epigastrium, and over the liver dulness amphoric and coin sounds can be obtained, and the tympanitic area travels round the chest as the patient is rolled over.

In only 2 of 76 cases was the liver dulness recorded to be lost, although this is usually considered a very constant sign of perforation. The special shape and signs of abdominal swellings in the different anatomical varieties of subphrenic abscess are recorded under that heading.

The *veins* of the abdominal wall were dilated in one case, and in another a *peritoneal rub* was heard and felt over the lower margin of a subphrenic abscess. *Ascites* was also present in one case, whilst *pus* was discharged *from the rectum* in a like number.

IV. *The Thoracic Signs and Symptoms of Subphrenic Abscesses.*—Thoracic signs were present in 56 of 76 cases of subphrenic abscess, and were only stated to be absent in 3 cases. In 41 cases the signs were present at only one base, but in 15 cases they were described at both. In 2 gastric cases the abscess was on one side and the basal signs on the other. The conditions indicated by these signs were those of compression, and inflammation of the base of the lung and pleura, namely :

(*a*) Displacement of the lung upwards, and obliteration of the lower pleural angle.
(*b*) Dry pleurisy.
(*c*) Pleural effusion.
(*d*) Compression of the lung.
(*e*) Consolidation of the lung.
(*f*) More or less bronchitis at the base.

The most frequent association of signs at the base was *dulness* (30 cases), *with diminution or absence of breath-sounds* (22 cases), *vocal resonance* (12 cases), and *tactile vocal fremitus* (16 cases).

Pleuritic friction-sounds were only recorded in 7 cases.

Dulness on percussion was also associated with *tubular breath-sounds* (9 cases) and *increased vocal resonance* (5 cases).

Râles, rhonchi, and crepitations, were recorded in 3 cases.

In 9 cases *tympanitic, amphoric, and coin sounds* were discovered at the base of the thorax, usually over the liver dulness, and were proved to be due to gas below the diaphragm. In 6 of these the *resonance shifted with position*.

The *gurgling* of gas and fluid was heard in one case, and a very *sudden spread of dulness* up the thorax was noted in another, and was proved at the operation to be due to a clear pleural effusion. The thorax was recorded to have *moved less on respiration* on the affected side in 12 cases.

Pericarditis was noted in only one case.

The *position of the heart apex-beat* was noted in 9 cases.

It was *displaced upwards*, but was very little if at all moved laterally in 7 cases.

In 2 cases in which pleural effusions complicated the subphrenic abscess, the apex-beat was considerably *displaced to the other side*.

Pain on deep inspiration and coughing was noted in 5 cases.

A severe *cough* was recorded in 17 cases. In 8 of these *pus* was expectorated, and in 2 cases *blood ;* whilst 1 patient expectorated *pus and flatus*, with collapse and profound mental anxiety.

In 5 cases it is definitely stated that *thoracic symptoms were present without expectoration*.

Dyspnœa was present in 7 cases, and varied from 32 to 48 breaths a minute. The average was 37.

The *alæ nasi* were seen in active movement in several cases.

V. *The Local Signs of Subphrenic Abscess.*—These local signs were found over the abscess and encroaching upon the thorax and abdomen, or both:

The abscess caused a visible *bulging of the thorax* or a definite *swelling in the abdomen* in no fewer than 46 of 76 cases. These swellings were located as follows:

Beneath the lower ribs, 11; in the right hypochondrium, 12; in the left hypochondrium, 15; in the epigastrium, 3; in the left loin, 3; in the right loin, 1; in the left abdomen, on a level with the umbilicus, 1.

In several cases the *circumference of the thorax* at the level of the ensiform cartilage was shown by the tape measure to be from 1 inch to 3 inches greater on the side of the abscess.

Deep tenderness was elicited in 25 cases on palpating deeply over the swelling. In 4, between the ribs; in 10, in the right hypochondrium; and in 11, in the left hypochondrium.

The *X rays* demonstrated the abscess as a dark shadow in the only case in which it was used.

Œdema was recorded over the abscess in 4 cases, and the *veins* were noted to be enlarged in 2.

The signs which indicated that the swelling contained gas, pus, or was formed of peritoneal adhesions, have been already described under the heading of Abdominal Signs. The most conclusive and certain sign, however, is needling; but this matter must be discussed at length.

The Exploratory Needle in Subphrenic Abscess.—The most careful examination often fails to make it certain whether a subphrenic abscess is present or not. If pus is then found below the diaphragm with the exploring needle, the case becomes clear, and the abscess can at once be opened. On the other hand, many authorities (Dr. Acland) are opposed to the use of the exploring needle because it is not free from the risk of infecting the pleura, and because, when the result of a puncture is negative, doubt and delay are only increased. This impeachment is confirmed by the experience of the hollow needle at the London Hospital. Of 76 cases, the aspirating needle was employed in 18, and in 11 of these the pus was not found. In 1 of these further measures were delayed for twelve days, and in another for five days, and in the others for various periods. In 1 case nothing was found on puncture with the needle at the right base, but the patient collapsed and died three hours later, and it was then found that 1½ pints of turbid offensive pus had passed through the puncture from the right subphrenic space into the pleura, and the lung was collapsed.

It is therefore clear that a promiscuous search for pus with an ineffective syringe is dangerous and deceptive.

On the other hand, the proper use of a good aspirating needle upon the operating-table, and under a full anæsthetic, is the most certain means of diagnosis we possess, and it has never failed or deceived in my own experience. The only safe rule in thoracic operations is to follow the needle, and it is useless to make a large opening and admit air to the pleura where the needle can demonstrate that no pus is present.

It may seem too elementary a matter to discuss the character of an efficient aspirating needle, but no other instrument is so often badly made and out of repair. The needle should be of large calibre, so that thick pus and lymph may be drawn through it. It must be at least 3 inches long, so that deep collections of pus may be reached, and well tempered, so that it may not break in the patient. I have removed two broken needles from the thoracic wall under these circumstances. The syringe must not leak either round the piston or where the needle is attached to it. The best way of testing this is to draw a little water into the syringe, and then plunge the end of the needle into a bar of soap, and draw the piston out to the full extent. It is easy then to see if the air enters at these points, and on releasing the piston it should fly back to the surface of the water, and no bubble of air should have appeared. If this precaution is not taken, and the syringe becomes filled with gas during its use, the operator cannot be certain that the gas entered from the point of the needle. Finally, the needle must be made of such material that it may be be boiled for aseptic reasons. Messrs. Krohne and Sesemann have made a very good needle which meets all these requirements.

During its use the syringe should be washed free of blood between the punctures in sterile water, and carbolic and other antiseptic lotions should be avoided, because they render clear serum and blood turbid, and simulate purulent fluids very closely. When air has filled the syringe whilst the thorax is being explored, the needle should be withdrawn in order to expel the air, and the syringe should never be unscrewed from the hollow needle whilst it is *in situ;* otherwise air may enter the pleura and the lung collapse. In one lamentable case of empyema with which I was acquainted, this accident happened to a medical man whilst he was puncturing the base of the other lung, which was also dull, and the patient did not survive.

When the signs and symptoms indicate the presence and location of pus below the diaphragm, the patient should be prepared for operation and fully anæsthetized, and only then should the needling be carried out. With the patient in the semi-prone position, I begin by passing the needle deeply into the tenth intercostal space in the scapular line, and then, if no pus is found, into the ninth, eighth, seventh, and sixth spaces in turn. I then search the spaces in the mid-axillary line, from below up. Several signs indicate that the needle has passed through the diaphragm. There is often the sensation of a second obstacle through which the needle is thrust. A deep puncture of, say, 3 inches in the lower intercostal spaces is certain to pass through the diaphragm. When the end of the needle is through the diaphragm, and the latter is not paralyzed by the inflammation, the barrel of the syringe moves up and down with respiration (Furbringer's sign). This is practically a very constant sign, although when the diaphragm is paralyzed the movement may be absent, or even reversed (Devic and Chalier). Finally, the character of the blood drawn into the syringe is an indication of where the point of the needle is. Blood from the liver is dark venous, and is often obtained in quantity from the hepatic sinuses. On the other hand, blood from the lung is foaming and scarlet, and is not obtained in large quantity unless a big vessel is entered, when hæmoptysis follows ; but this is a very rare occurrence, and is practically never a serious matter.

The needle should be passed in to its full depth of about 3 inches, and then slowly withdrawn whilst suction is maintained. Timidity may lead the operator to only enter the pleura, from which he may obtain nothing or only clear serum. Thus, in the 11 cases at the London Hospital in which pus was present below the diaphragm, and the hollow needle failed to find it, in 5 nothing was withdrawn, in 4 clear serum was obtained, and in 2 blood-stained serum.

When pus is found, and the needle is known to be below the diaphragm, the diagnosis of subphrenic or hepatic abscess is certain. When different fluids are withdrawn at different depths or at separate punctures, the diagnosis is also highly probable. Thus clear serum and pus may be associated, or foul gas and clear serum, or even foul pus and sweet pus.

Needling should never be abandoned until it is certain that no pus is present; and it may be added that, although the method is so free from risk when practised through the thoracic wall, as recommended above, it is never safe to puncture abdominal swellings across the peritoneum and through the abdominal wall, because they afterwards leak into the peritoneum. Thus a tuberculous pyonephrosis which was punctured in the loin led to acute general peritonitis. When pus has been found, the rib below should be resected, and after the diaphragm has been sewn to the intercostal muscles I usually make another puncture before incising it.

The Operations for Opening Subphrenic Abscesses.—The methods employed to drain subphrenic abscesses may be arranged as follows, and I have added the number of cases treated by each method among the 73 operations performed on 64 cases at the London Hospital (1900 to 1907):

		Operations.	Deaths.
A.—Thoracic.			
Posterior transpleural (scapular line)...	Right	11	3
	Left	3	1
Subpleural ...	Right	0	0
	Left	1	0
Anterior transpleural (nipple line)	Right	0	0
	Left	1	1
Subpleural ...	Right	2	1
	Left	1	0
Lateral transpleural (axillary) ...	Right	1	1
	Left	0	0
Subpleural ...	Right	3	2
	Left	0	0
B.—Abdominal.			
Median ...	—	5	1
Hypochondriac or epigastric ...	Right	17	9
	Left	17	5
Loin ...	Right	6	2
	Left	5	1

Speaking in a general manner, posterior methods of drainage are more favourable than anterior. In the above series 26 posterior operations were performed, with 7 deaths (27 per cent.); 43 anterior operations, with 17 deaths (39·5 per cent.); and 4 lateral, with 3 deaths (75 per cent.).

I have performed 24 operations on 21 patients for subphrenic abscesses: 15 of these were posterior, with 2 deaths (13·3 per cent.); 9 were anterior, with 2 deaths (22·2 per cent.). The posterior routes should always be adopted when it is possible.

The Posterior Transpleural Thoracic Operation.—As far as possible this operation is carried out in the line of the angle of the scapula, with the patient semi-prone. The exploring needle is inserted through the intercostal spaces, in turn from below up, beginning at the tenth space. When pus is found, 3 inches of the rib below is resected, whilst an assistant presses up the liver on the right side to obliterate the pleural cavity, should it be open. The diaphragm is then firmly fixed to the intercostal muscles with three or four interrupted catgut sutures inserted by a sharply curved needle in a handle. Pus is then once more found with the exploring needle before the diaphragm is incised, and a finger passed into the abscess cavity. This finger should hook the diaphragm well up against the thoracic wall whilst the pus escapes, and before it is withdrawn the diaphragm and intercostal muscles should be sewn together by a continuous catgut suture. This should be done whether adhesions are present or not, for, as the abscess emptied and the diaphragm descended, the adhesions gave way in 5 cases in my series, and pneumothorax resulted, in 2 cases at the operation, and in 3 cases after a day or two. Silk should not be employed for these sutures, because in one case a sinus persisted for two and a half years, but closed as soon as a stout silk suture was discharged. As large a drainage-tube as is convenient should be employed, and irrigation, suction, and position may be employed to clear the cavity after a day or two.

No rib higher than the seventh should be resected, for great difficulty will then be found in bringing the diaphragm up to the thoracic wall. The exploring finger in a right anterior intraperitoneal subphrenic abscess can often feel the upper surface of the liver deeply cupped below, whilst above the diaphragm is thrown into great folds which contract in a futile manner at each inspiration.

The Posterior Subpleural Thoracic Operation.—When the tenth or eleventh ribs have been resected in the line of the angle of the scapula, it is stated by Elsberg that the reflection of the pleura may be recognized and pushed up before the diaphragm is incised. This method is, however, needless if the above precautions are taken to prevent air and pus from entering the pleura (see also Figs. 64 and 65).

The Anterior Subpleural Thoracic Operation.—Landau has pointed out that in the mammary line about 2 inches intervenes between the costal margin and the reflection of the pleura. If the costal cartilages are resected over this space, an incision may be made through the diaphragm, below the pleura and above the peritoneal adhesions shutting in the abscess. This method is especially adapted to left anterior perigastric abscesses. It may also be used at other positions on the costal margin, but the space is narrower there (Figs. 64 and 65). The series of 76 cases at the London Hospital contained 8 such operations, with 5 deaths (62·5 per cent.), and in several of these the pleura was opened none the less. The operation is moreover anterior, and drains badly.

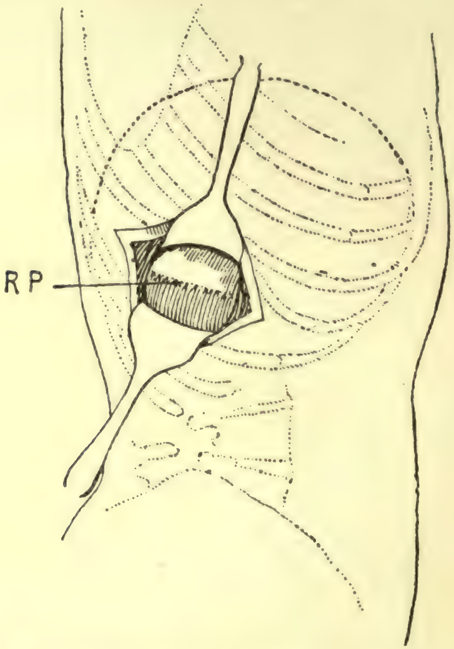

FIG. 64.—THE REFLEXION OF THE PLEURA, RP, IN A LATERAL THORACIC SUBPLEURAL OPERATION. (From Kelly.)

The writer does not favour the lateral operation, nor in his experience is the subpleural space so liberal as is indicated here in the ninth intercostal space.

The Median Abdominal Operation.—This route is usually adopted when a strictly median swelling is pointing in the epigastrium, and when the exploring needle has shown that the pus cannot be reached from behind. When the swelling is due to a right extraperitoneal or hepatic abscess tracking forward between the layers of the falciform ligament, it is possible to open the abscess without wounding the peritoneum if the incision is strictly median (*vide* Fig. 62), and the opening is deepened at the upper end immediately below the ensiform process. I have operated on two

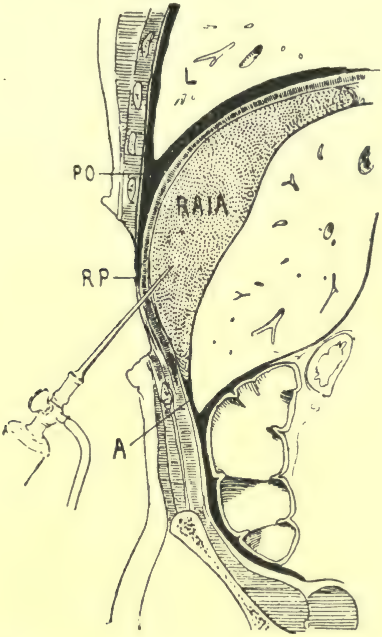

FIG. 65.—"THE LATERAL SUBPLEURAL THORACIC OPERATION." (From Kelly's "Appendicitis.")

RAIA is a right anterior intraperitoneal abscess, which, having localized below the diaphragm, has extended down as low as the tenth space. The pleura, PO, is obliterated below, and the lung, L, is pushed up. The reflexion of the pleura, RP, is seen below. The ninth or tenth rib has been resected, and the exploring needle is being inserted below the pleura. Below, the adhesions, A, of the liver to the diaphragm are seen shutting off the abscess. These adhesions might in a recent case be separated by a finger passed upwards through an oblique loin incision, but after ten days the liver would probably be torn. This figure illustrates the advantages of waiting a reasonable time for a subphrenic abscess to develop.

such cases successfully. An abscess occupying the small sac of the peritoneum may project forwards between the liver and the stomach. The general peritoneal cavity is then opened, and the bulging small omentum is sewn to the parietal peritoneum before it is incised, and a large drainage-tube is inserted. I have drained one such case, and at the autopsy, three weeks later, the cavity was found empty and nearly closed. Death was due to suppurative cholangitis and pyæmia.

Right and Left Hypochondriac and Epigastric Operations.—These operations are adapted to the drainage of those right and left anterior intraperitoneal abscesses which present in the epigastrium, and give rise to the triangular swelling which is described under the headings of these special forms of subphrenic abscess. Even when an anterior swelling is present, I prefer the posterior transpleural route if the exploring needle finds pus behind.

When it has been decided to open the abscess from in front, a vertical incision is made well within the triangle. It must not encroach at all on the band of adhesions below, lest the pus or gastric juice should be admitted to the general peritoneal cavity. The incision is therefore deepened at its upper end, and I prefer to incise the peritoneum in the angle between the costal margin and the ensiform process.

The Loin Operation for Subphrenic Abscess. — The right loin operation is adapted to the drainage of the early and diffuse infections of the subhepatic pouch and the right anterior intraperitoneal fossa when the liver is not firmly adherent to the diaphragm. At all stages it is the method to be selected for the drainage of the subhepatic pouch. On the left side it is employed alone for the diffuse stage of peritonitis. In some cases, where a very large left anterior intraperitoneal subphrenic abscess has been opened in the left epigastrium, and projects sufficiently below the ribs, it may be counter-opened in the left loin.

The incision is an oblique one downwards and forwards, far back, and high up in the loin. When the peritoneum is opened, should no adhesions be present, the general peritoneal cavity is packed off with gauze below and in front. The index-finger then feels for the margin of the liver on the right side, and is passed transversely below it into the subhepatic pouch, into which a drainage-tube can then be introduced. Should the signs indicate the presence of pus in the right anterior intraperitoneal subphrenic space, and in all doubtful cases, the finger is then insinuated between the liver margin and the diaphragm separating the adhesions. A drainage-tube is then introduced up the side of the finger, and pushed up under the dome of the diaphragm as far as it will go—often a distance of 6 or 7 inches. Should a lumbar abscess be present, a tube may be passed down the loin. In one case I opened and drained a large perinephritic and retrohepatic abscess from the posterior angle of this wound in addition to intraperitoneal abscesses in front. The procedure is very similar upon the left side, but here the exploring finger feels for the tail of the spleen, and the tube is pushed up into the left anterior intraperitoneal fossa. When the abscess is retroperitoneal and left, it usually projects in the loin, and an incision is made over its most prominent part and a tube passed up under the diaphragm as before.

The Methods of Irrigation Suction and Position in Dependent Cavities.—When it has been found necessary to open a subphrenic abscess from the front, the cavity is usually dependent, so that gravity does not insure complete drainage. The methods which should then be adopted are as follows: The largest size of drainage-tube ($\frac{3}{4}$ inch) should be passed to the very bottom of the cavity to act as a well for the pus. A long strip of gauze may be passed down the inside of this to soak up the fluid by capillary action into the dressings. Twice a day the cavity should be washed clean with sterile water or very weak carbolic (1 in 100), through an elastic catheter passed down the inside of the drainage-tube to the bottom of the cavity. The cavity should then be sucked dry by the same catheter applied to a syringe. At intervals during the day the patient should sit up and lie semi-prone on the opposite side to the abscess cavity, so that the contents may gravitate along the tube. With these precautions the temperature will remain normal, the abscess will rapidly close, and very little tendency will be shown by the abscess to pocket and track, although a constant watch should be kept on neighbouring cavities and fossæ.

The Mortality of Subphrenic Abscesses.—Of the 76 consecutive cases which make up this series, 40 lived and 36 died, a mortality of 47·4 per cent.; 12 cases were not operated upon, and none of these survived, a mortality of 100 per cent.; 64 cases were operated upon (by 73 operations) of the series of 76 consecutive cases, and of these 24 died, making a mortality of 37·5 per cent. These operations were divided up as follows: 26 posterior operations with 7 deaths, 27 per cent.; 43 anterior operations with 17 deaths, 39·5 per cent.; and 4 lateral operations with 3 deaths, 75 per cent.

Posterior operations are therefore more favourable. Of my own series of 21 consecutive cases submitted to 24 operations, 4 died, 19 per cent.; 15 of these operations were posterior, with 2 deaths, 13·3 per cent.; and 9 were anterior, with 2 deaths, 22·2 per cent.

I have analyzed the cause of death in 36 cases, and have found that it was unavoidable in 12 cases, and might have been avoided in 24 cases. The 12 unavoidable causes of death were—5 multiple abscesses of liver, 2 cancers, 2 lung complications, 2 hour-glass stomachs, and 1 general peritonitis. It has been truly said that "wise men learn by others' experience," and it will therefore not be time wasted to consider in detail the 24 deaths which may be classed as avoidable. In 12 cases the condition was not diagnosed, and no operation was performed. For the most part these cases died in the medical wards. In 8 cases an anterior incision was made into the abscess, which was inefficiently drained. Other loculi of pus were left undrained in several cases. By a fuller knowledge of the subphrenic spaces, and further incisions, these cases might have been saved. In 2 cases the transpleural method was adopted during the acute and diffuse stage, when the loin incision was indicated. The pus was not efficiently drained, and in one case the lung collapsed. In 1 case a puncture by a needle failed to find pus, but admitted more than a pint of pus and gas to the pleura. In one case an epigastric incision was prolonged downwards beyond the line of adhesions, so that, when the abscess which communicated with the stomach was opened in the upper part of the incision, it gravitated into the general peritoneal cavity in the lower angle of the wound.

In short, half the avoidable deaths may be attributed to the fact that the condition was not diagnosed at all; the other half, to the absence of exact and detailed knowledge of the localization and character of subphrenic abscesses on the part of the surgeons when they were planning and executing their operations.

With a perfect opportunity, knowledge, and technique, these 76 cases might have been treated with an ideal mortality of no more than 16 per cent.

The diagrams illustrating this paper were drawn by Dr. Stanley Beale, to whom my best thanks are due for their skill and lucidity.

APPENDIX

Anatomical Varieties.

	No. of Cases.	Simple.	Compound.
A.—INTRAPERITONEAL.			
I. Right:			
(*a*) Anterior	27	11	16
(*b*) Posterior	10	0	10
II. Left:			
(*a*) Anterior	30	26	4
(*b*) Posterior	3	1	2
B.—EXTRAPERITONEAL.			
I. Right	19	13	6
II. Left	4	4	0
Not classified	2	—	—

Simple = One cavity only involved.
Compound = Two or more cavities involved.

Anatomical Varieties according to Causes.

	Gastric Ulcer.	Appendicitis.	Hepatic Abscesses.	Duodenal Ulcer.
Intraperitoneal, right anterior ...	4	10	7	1
Intraperitoneal, right posterior ...	1	6	1	2
Intraperitoneal, left anterior ...	16	1	1	1
Intraperitoneal, left posterior ...	2	0	0	0
Extraperitoneal, right	0	1	15	2
Extraperitoneal, left	1	0	0	0

Etiology.

	Cases.
Gastric ulcer	21
Gastric cancer	2
Appendicitis	12
Liver: Suppurating hydatid	8
Liver: Tropical abscess	6
Liver: Abscess	1
Pylephlebitis	1
Suppurative cholangitis	1

Etiology (continued).

	Cases.
Duodenal ulcer (anterior)	3
Duodenal ulcer (posterior)	2
Splenic abscess	3
Pyæmia	3
Parturition	3
Thoracic pneumonia	1
Thoracic bronchiectasis	1
Thoracic empyema	1
Periostitis of vertebræ	2
Operations, resections	2
Pancreas cancer	1
Kidney, cystic	1
Intestine ruptured	1
Typhoid	1
Gall-stones	1
Pyosalpinx	1
No cause found	2

Age of 75 Cases of Subphrenic Abscess arranged in Decades.

Age.	Cases.
0–10	6
10–20	5
20–30	29
30–40	15
40–50	13
50–60	6
60–70	1

Sex of 76 Cases of Subphrenic Abscess.

	Cases.	Per Cent.
Male	43	56·6
Female	33	43·4
Total	76	100

The Spontaneous Rupture of 23 Cases of 76 Subphrenic Abscess.

Into—	Cases.
A bronchus	4
The pleura, right	2
The pleura, left	3
The general peritoneum	1
The stomach	8
The intestine	1
The colon	2
The skin—(*a*) umbilicus	1
(*b*) right hypochondrium	1
	23

Micro-Organisms found in Pus of Subphrenic Abscess.

	Cases.
Sterile	3
B. coli communis	3
Staphylococcus aureus	2
Streptococcus	1
Pneumococcus	1
B. pyocyaneus	1
B. typhosus	1
Total number examined	12

Onset in 76 Cases of Subphrenic Abscess.

Acute	40 cases = 62·6 per cent.
Subacute Chronic	36 cases = 47·4 per cent.
Gastric ulcer (21 cases):	
Acute	15
Subacute Chronic	6
Gastric cancer (2 cases):	
Acute	0
Subacute Chronic	2
Appendicitis (12 cases):	
Acute	12
Subacute Chronic	0
Hydatid of liver (8 cases):	
Acute	2
Subacute Chronic	6
Hepatic abscesses (7 cases):	
Acute	1
Subacute Chronic	6
Duodenal ulcer (5 cases):	
Anterior (= Acute)	3
Posterior (= Subacute)	2
Splenic abscess (3 cases):	
Subacute	3

The Significance of Rigors in Subphrenic Abscess.

	Cases.
Number recorded	10
Died	6
Pyæmias (2 chronic ones lived)	5
Communicated with stomach	2
Suppurative cholangitis	1
Acute periostitis	1

The Use of the Aspirating Needle in 18 *Cases of Subphrenic Abscess (Some Several Times).*

	Cases.
Failed to diagnose	11
Pus	7
Foul pus	2
Anchovy pus	1
Pus and serum	1
Clear serum	4
Blood-stained serum	2
Gas	2

Mortality of 76 *Cases of Subphrenic Abscess.*

	Deaths.		Per Cent.
76 cases	36	=	47·4
12 cases no operation	12	=	100·0
64 cases operated on	24	=	37·0
21 cases operated on by author	4	=	19·0

ANALYSIS OF 36 DEATHS.

A.—Unavoidable.—12 deaths = about 16 per cent.

	Cases.
Multiple abscesses of liver	5
Cancers	2
Hour-glass stomach	2
Lung complications	2
General peritonitis	1

B.—Avoidable (probably).—24 deaths.

	Cases.
Not operated on	12
Anterior operation; other pouches left	8
Transpleural operation in diffuse stage	2
General peritoneum opened by anterior incision	1
Inefficient needling	1

The ideal mortality of subphrenic abscess = about 16 per cent.

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